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BUREAU OF HYGIENE AND TROPICAL DISEASES.

TROPICAL DISEASES BULLETIN.

Vol. 38.] 1941. [No. 12.

FEVERS OF THE TYPHUS GROUP AND OTHER FEVERS.

Précis of Abstracts in this Section.

General.—In the Annual Report of the League of Nations, Eastern Bureau (p. 679) there is mention of the *Proteus OXK* form in Sumatra and in India, and of the epidemic of the *Proteus OX19* form in Shanghai.

Singer (p. 679) reports experiments in which limited multiplication of the Rickettsiae of typhus, Rocky Mountain fever and Q fever, appeared to occur in cell-free media.

Reynes (p. 680) states that sera of patients suffering from fevers of the typhus group may become temporarily or persistently positive to several of the recognized tests for syphilis.

Goyal (p. 680) has found Rickettsiae, non-pathogenic for man, in the brains of rats in Calcutta.

Louse- and flea-borne.—Liu *et al.* (p. 681) show that although in normal mice epidemic European typhus exists as an inapparent infection only, a fatal infection may be induced by irradiating the animals and administering heavy infecting doses by the abdominal route.

Dyer (p. 681) discusses the prevention of murine and of epidemic typhus.

Meleney (p. 681) discusses the recent extension of endemic typhus in the United States, which has been especially evident in cities and towns, but has also occurred in rural areas. Silva and Ochoa (p. 682) have isolated a murine strain from rats in Zacoalco, Mexico ; a similar strain had previously been isolated from a human patient. It is claimed that in the high plateaux the murine form is spread from man to man by lice. [A similar claim has been made in Shanghai by Raynal.]

Bengtson (p. 682) discusses the complement fixation test in endemic typhus.

Tick-borne.—Brigham and Watt (p. 682) have recovered a highly virulent strain of Rocky Mountain fever virus from *Dermacentor variabilis* in Georgia. In general, the strains found in eastern U.S.A. are less virulent than those in the west. Hassin (p. 683) gives a description of the histo-pathological appearances in a case of Rocky Mountain fever in which there was non-suppurative meningo-encephalitis. Ludlam *et al.* (p. 683) record a case of probable tick-borne typhus, which occurred at a Scottish port. It is thought that the patient was infected by a tick brought in cargo from S. America.

Mite-borne.—VAN DER SCHROEFF (p. 684) describes an outbreak of mite fever and tropical typhus, in N. Sumatra, among labourers engaged in clearing jungle. He contends that the diseases are identical, the absence of primary ulcer in tropical typhus being due, possibly, to transmission by ticks which, in the act of biting, penetrate the tissues more deeply than mites. LEIMENA (p. 685) reports a case of the *Proteus OXK* type of fever from Java, where the disease is but rarely seen. MAY (p. 685) records the *Proteus OXK* form from Papua. KAWAHIGASI (p. 685) reports on mite-borne typhus in Formosa. KOUWENAAR and ESSEVELD (p. 686) note that Sumatran mite fever produces immunity in guineapigs for at least 13 months, but that convalescent serum has no protective or curative effect.

Vaccination.—DURAND and GIROUD (p. 686) describe the preparation of vaccine against epidemic typhus by the use of the lungs of rabbits infected by the tracheal route. Rabbits are not easy to infect in this way, but by the use of a suitable technique a heavy infection can be produced, which provides a vaccine of good immunizing power.

FINLAYSON and GROBLER (p. 687) show that three doses of vaccine give better protection than one to guineapigs, but state that alum-treated vaccines protect 80 per cent. of animals even when only one dose is given. RUIZ-CASTANEDA (p. 687) shows that vaccine of a murine strain will protect guineapigs against infection with epidemic typhus.

PARKER (p. 688) shows that vaccine prepared from infected ticks gives good protection against Rocky Mountain fever for one year. In areas of high virulence it protects against death but not much against infection ; in areas of low virulence it is effective against infection.

Q fever.—BURNET and FREEMAN (p. 688) confirm previous findings that the American and Australian strains of Q fever virus are immunologically identical but that the former is the more virulent. The virus, mixed with immune serum, or treated with lauryl sulphate, gives immunity but no fever on injection into guineapigs. Killed or formalin-treated Rickettsiae produce immunity. They (p. 689) describe the cultivation of the American strain of Q fever virus on chicken embryos. Virulence is increased after such culture and is comparable with that which occurs in the tick with the virus of Rocky Mountain fever ; in both cases it is thought that the fact that no antibodies are formed may be the explanation of this increased virulence.

DAVIS (p. 689) shows that *Rickettsia diaporica* may persist in the tissues of *Ornithodorus turicata* for as long as 1,001 days. The ticks do not apparently transmit the organisms by bite, or to their progeny, but the excreta are infective. SMITH (p. 690) quotes evidence which shows that in Australia *Rhipicephalus sanguineus* must be regarded as a potential vector of Q fever.

BENGTSON (p. 690) discusses active and passive immunity in relation to Q fever in experimental animals.

She (p. 690) refers to work on the complement fixation reaction in Q fever, which is specific for that infection.

HORNIBROOK *et al.* (p. 691) describe an outbreak of Q fever in employees of the National Institute of Health, Washington. The source of infection was not discovered, but the organism was under investigation in the building at the time and it is probable that the outbreak was due to inhalation of infected dust. All the patients showed X-ray evidence of patchy central pneumonia. HESDORFFER and DUFFALO

(p. 691) report a case of Q fever, with patchy pneumonitis, in Montana. The Weil-Felix test was negative, but *R. diaporica* was agglutinated.

Sandfly fever, etc.—WALKER and DODS (p. 692) give an account of sandfly fever in Palestine, where there was an epidemic in 1940. The disease usually lasts more than three days, and secondary rise of fever occurs in some cases. There is apparently no lasting immunity. A sensation of constriction round the thorax and a crop of vesicles on the palate were constantly present. PEARSON (p. 693) shows that sandfly fever may be one of the causes of benign lymphocytic meningitis.

TOPPING *et al.* (p. 694) report on cases of Colorado tick fever, probably due to a virus transmitted by *Dermacentor andersoni*. In comment, MEGAW notes the similarity between this disease and dengue and suggests the name tick dengue.

Bartonellosis.—PATIÑO-CAMARGO (p. 695) reports a case of Guáitara fever (bartonellosis) in Bogotá. GEIMAN (p. 695) gives details of the preparation of three media for the cultivation of *Bartonella bacilliformis*; growth with any of these is luxuriant enough to permit immunological and other investigations. *C. W.*

DARRIBA (A. Rodríguez). Rickettsiosis humanas. [**Human Rickettsial Diseases.**]—*Revista Clínica Española.* Madrid. 1941. Feb. 1. Vol. 2. No. 2. pp. 113–128.

This is a general résumé of present knowledge of fevers of the typhus group. No new work is reported. A table of deaths due to epidemic typhus in Spain from 1911 to 1930 is given. *C. W.*

LEAGUE OF NATIONS. HEALTH ORGANISATION. EASTERN BUREAU, SINGAPORE. ANNUAL REPORT FOR 1940. pp. 64–68. With 3 graphs.—(J) **Typhus in Countries.** (K) **Typhus in Sea and Air Ports.**

In Northern Sumatra about 500 cases of typhus have been reported each year for the last 10 years; of an average of 490 cases annually during the last 5 years, 425 were of the *Proteus XK* type. The distinction between scrub typhus and tsutsugamushi, that an ulcer is present in the latter but not in the former, is not now held to be valid. In India there is evidence of seasonal variation of the *XK* type, which is most common in August and September, towards the end of the monsoon. This seasonal incidence is probably connected with the biology of the vectors.

Typhus is prevalent in Shanghai, dating from the influx of refugees in 1937. RAYNAL concludes that there is a form intermediate between classical louse-borne and murine typhus, and that the latter may be transmitted by the louse. The epidemics reach their peak between March and July, when climatic conditions are moderate. *C. W.*

SINGER (E.). **Experiments on the Survival of Rickettsiae in Cell-free Media.**—*Australian Jl. Experim. Biol. & Med. Sci.* 1941. June. Vol. 19. Pt. 2. pp. 123–124.

Two sets of experiments are briefly described in which limited multiplication of Rickettsiae appears to have occurred in cell-free media.

The first set was carried out "some years ago" when the Rickettsiae of epidemic typhus and Rocky Mountain spotted fever were cultivated

up to from 3 to 5 passages in a medium consisting of equal parts of guineapig serum, Tyrode's solution and 1 per cent. glutathione (G.S.H.) solution. The Rickettsiae were first adapted to tissue culture conditions by cultivation on the tunica vaginalis of a guineapig in serum-Tyrode mixtures.

The second set of experiments was done recently on the *R. burneti* of Q fever. Preliminary cultivations were carried out with weekly passages in a medium consisting of serum-Tyrode mixtures to which guineapig spleen was added.

Success was obtained only in one in ten of the first transfers through this medium but from the third transfer onwards "takes" were secured in about 3 of every 5 cultures up to the 19th transfer.

Material from transfers later than the third was used in attempts to grow the Rickettsiae on various cell-free media. Evidence of multiplication of the virus was obtained only with the following :—

(*a*) Up to the second transfer, with serum-Tyrode mixtures incubated for a week with guineapig spleen.

(*b*) Up to the third transfer, with a serum-Tyrode mixture separated by a semi-permeable cellophane membrane (treated by Seymour's method—*Jl. Biol. Chem.*, 1940, Vol. 134, p. 701) from a serum-Tyrode mixture incubated for one week with guineapig spleen.

(*c*) Also up to the third transfer with a serum-Tyrode mixture separated by a similar membrane from fresh Tyrode extract of spleen.

John W. D. Megaw.

REYNES (V.). Les réactions de diagnostic de la syphilis au cours des fièvres typho-exanthématiques. [**Serum Reactions of Syphilis in Fevers of the Typhus Group.**]—*Bull. Soc. Path. Exot.* 1940. Vol. 34. Nos. 1–3. pp. 51–57.

At the Pasteur Institute of Saigon, sera from seven cases of tropical typhus which reacted to *Proteus OXK* were repeatedly examined by several of the recognized serum tests for syphilis.

The usual result was that reactions which were negative in the early stage of the disease soon became temporarily or persistently positive. In one case the reaction was positive at the outset, but became negative during the short period when the reaction to *OXK* was at its highest ; afterwards it became more strongly positive than it had been at the beginning of the illness.

The sera of seven cases of endemic typhus (reacting to *Proteus OX19*) were examined in the same way ; five of these remained negative throughout and two became positive for a few days.

The only test that gave consistent results was the Kahn reaction ; the Calmette-Massol, Hecht-Mutermilch and Meinicke tests appear from the tables to have given such irregular responses as to be of doubtful significance. *J. W. D. M.*

GOYAL (R. K.). **The Presence of an Enzootic of Rickettsial Infection in Wild Rats of Calcutta.**—*Indian Med. Gaz.* 1941. Feb. Vol. 76. No. 2. pp. 84–86.

From January 1937 to December 1938, 100 rats were caught in various districts in Calcutta ; these were killed by drowning and the brains were extracted and incubated ; bacteria-free brains were emulsified and injected into male guineapigs. Seventy-two per cent.

gave a positive reaction, a mild fever lasting three or four days with some thickening of the tunica vaginalis; smears taken from the tunica, spleen and brain showed the presence of both extra- and intracellular pleomorphic Rickettsial bodies. There was no positive Weil-Felix reaction in any of the guineapigs, and rabbits inoculated with the virus showed no agglutinins for any of the strains of *Proteus X*. An emulsion of the brain and tunica of an infected guineapig was inoculated into two volunteers, there was no reaction of any kind. The author concludes that the rat strain of virus was non-pathogenic for man. Three hundred other rats were killed and examined during the period September 1939 to July 1940, but none was found to be infected. *D. Harvey.*

LIU (P. Y.), SNYDER (John C.) & ENDERS (John F.). **Fatal Infection of Irradiated White Mice with European Typhus by the Intra-Abdominal Route.**—*Jl. Experim. Med.* 1941. May 1. Vol. 73. No. 5. pp. 669–680. With 1 plate.

In normal mice European typhus exists as an inapparent infection in which the virus disappears after three passages and Rickettsiae cannot be demonstrated. Experiments were carried out and are described in this paper which show that if mice are irradiated and then given considerable doses of the virus of epidemic typhus by the abdominal route, 100 per cent. of the animals become infected and die and very numerous Rickettsiae are found in the peritoneal exudate and in the spleen and other organs of the body; by this means mice can be utilized as experimental animals in place of guineapigs and without the risk of laboratory infection which arises when the nasal route of infection is employed. Normal mice failed to show any signs of infection when inoculated with material collected from the irradiated animals. This method might be employed for general experimental work with this virus and also for the preparation of vaccines. *D. H.*

DYER (R. E.). **The Charles Franklin Craig Lecture for 1940. The Control of Typhus Fever.**—*Amer. Jl. Trop. Med.* 1941. Mar. Vol. 21. No. 2. pp. 163–183. With 1 map & 1 chart. [44 refs.]

This lecture was delivered at the 36th Annual Meeting of the American Society of Tropical Medicine at Louisville, Kentucky, on November 12th, 1940. The paper is divided into two parts, one dealing with the control of endemic typhus as it occurs in the United States, and the other dealing with the control of epidemic typhus as it has occurred in Europe and Asia and may occur again under war conditions. As regards endemic typhus the problem is one dealing with rat eradication, or at least reduction of numbers, and also rat protection of houses and stores. Immunization has no place in dealing with endemic typhus. On the other hand epidemic louse-borne typhus must be dealt with by thorough and effective delousing of the stricken population and in addition active immunization by means of killed Rickettsial vaccines. *D. H.*

MELENEY (Henry E.). **Recent Extension of Endemic Typhus Fever in the Southern United States.**—*Amer. Jl. Public Health.* 1941. Mar. Vol. 31. No. 3. pp. 219–227. With 5 maps. [20 refs.]

A paper read before the Epidemiological Section of the American Public Health Association.

The incidence of endemic typhus has increased rapidly in the Atlantic and Gulf States from North Carolina to Texas and also in Tennessee during the last decade. There has been a similar but smaller increase in Southern California. In all of these States except Alabama more cases were reported in 1939 than in any other year. This increase has consisted of a higher incidence in most of the large cities, where the diesase was first discovered; and also in extension to other cities and towns and to rural areas. The highest case rates appear to be in the small towns. In order to control endemic typhus where it is now present, it will be necessary to instigate more vigorous rat eradication programmes than have been carried out in the past, and in order to prevent its spread to new areas it is advisable for health departments in unaffected localities to begin similar programmes. *D. H.*

SILVA (Roberto) & OCHOA (R. Reyes). El tifo de Zacoalco. II.—Aislamiento del virus de ratas salvajes. [**Isolation of Typhus Virus from Wild Rats in Zacoalco.**]—*Medicina.* Mexico. 1941. Apr. 10. Vol. 21. No. 385. pp. 146–148. English summary.

"There is an exanthematic infection at Zacoalco, Jalisco, which was suspected of being typhus fever by Diaz in 1936 and confirmed by Castañeda in 1939, who isolated a strain from a patient, this strain had the characteristics of murine typhus. We have corroborated this by the isolation, from the brains of rats captured at Zacoalco, of a virus which has the same properties as that obtained by Castañeda. So far, this is the first zone of the Republic of Mexico where murine typhus has been found to occur in pure form due to the scarcity of lice.

"In the high plateaus the murine infection is spread from man to man by means of *Pediculus vestimenti.*" [See this *Bulletin,* 1939, Vol. 36, p. 994, and, for a similar transmission of murine infection in Shanghai, this *Bulletin,* 1940, Vol. 37, p. 256.]

BENGTSON (Ida A.). **Complement Fixation in Endemic Typhus Fever.** —*Public Health Rep.* 1941. Mar. 28. Vol. 56. Nov. 13. pp. 649–653.

The results obtained in the tests described indicate the usefulness of complement fixation in detecting recent and also past infection with endemic typhus virus. The antigen used was a suspension of Rickettsiae from the lungs of mice or the yolk sac of embryos infected with endemic strains. The sensitivity of the test is indicated by the fact that positive results were obtained with sera from cases in which infection had occurred as long ago as nine years and as recently as seven days. No fixation was obtained with sera from cases of Rocky Mountain fever or from Q fever cases ; this shows the specificity of the reaction.

D. H.

BRIGHAM (George D.) & WATT (James). **Highly Virulent Strains of Rocky Mountain Spotted Fever Virus isolated from Ticks (*D. variabilis*) in Georgia.**—*Public Health Rep.* 1940. Nov. 15. Vol. 55. No. 46. pp. 2125–2126.

"The presence of typical scrotal lesions, a shorter average incubation period, and a high fatality rate among the guineapigs inoculated with strains of Rocky Mountain spotted fever virus recovered in western

United States has served in the past to distinguish these strains from those isolated in the eastern part of the country." Recently, however, TOPPING and DYER [this *Bulletin*, 1940, Vol. 37, p. 843] have recovered a highly virulent strain from a human patient in the East. The present authors now report the recovery of two highly virulent strains from dog ticks (*Dermacentor variabilis*) in Georgia ; the owner of the dog from which one of the ticks was obtained was suffering from a typical attack of Rocky Mountain fever. The scrotal reaction, observed in guineapigs inoculated with one of the two (identical) strains is described :—

"It appears a few days after the onset of fever as a diffuse erythema followed by a macular rash which becomes purpuric within 24 hours. There is a slight edema of the cutaneous structures. The lesions may stop at this point or progress to the stage of necrosis and sloughing of the superficial layers of the skin. Healing usually begins before the slough is complete and progresses from both the bottom and sides of the ulcer. Characteristic scarring results."

The strains isolated were shown to be those of Rocky Mountain fever virus by the clinical picture in guineapigs and by cross immunity tests ; there was no cross immunity with strains of endemic typhus virus. Tables are given showing the similarity in incubation period and scrotal reaction between the tick strain and a western strain, but it was noticed that the fatality rate in guineapigs with the latter was higher than with the former. *C. W.*

HASSIN (George B.). **Cerebral Changes in Rocky Mountain Spotted Fever.**—*Arch. Neurol. & Psychiatrics.* 1940. Dec. Vol. 44. No. 6. pp. 1290–1295. With 1 fig.

A girl, 11 years of age, died on the eleventh day after she had been bitten by a tick and on the sixth day of fever ; she was admitted to hospital on that day, comatose, and died in half an hour.

A very careful histopathological study of the brain was made by the author, who has already carried out similar studies in fatal cases of typhus and other types of encephalitis.

The histological picture in this case was that of aseptic meningitis which was in evidence over the cerebral convexity and the base of the brain including the cerebellum. An unusual feature was the presence of histiocytes which were packed with lipoids and formed distinct foci. Nodes or nodules, dense aggregations of nuclei, were scattered throughout the brain including the medulla and the portion of the spinal cord which was examined. The elements which made up a nodule were practically always naked nuclei, only a few of which possessed any cytoplasm ; they were probably the nuclei of glia cells. The changes may be summed up as those of non-suppurative meningo-encephalitis and on the whole were analogous to those of typhus fever encephalitis.

D. H.

LUDLAM (G. B.), BATCHELOR (I. R. C.) & RHODES (A. J.). **Possible Case of Tick-borne Typhus in a Scottish Port.**—*Lancet.* 1941. Sept. 6. pp. 278–279. [11 refs.]

This is a remarkable case. The patient was a dock worker in a Scottish port ; he had been employed for 10 days in unloading a cargo of hides from a rat-infested ship from the Argentine.

The onset, on October 17th, 1940, was about 12 days after the completion of the work. By the seventh day the patient noticed a rash, he came

under observation on the ninth day when the rash was seen to be reddish-brown, macular, slightly papular and associated with petechiae. The trunk and limbs were affected, but the palms and soles showed no rash. The fever lasted about 21 days. The serum reactions were—positive to *Proteus OX2*, up to a titre of 1–480, but negative to *OX19*, and *OXK* throughout. Positive to *Bact. paratyphosum B* (*H*) up to the same titre, but negative to *Bact. paratyphosum B* (*O*), to *Bact. paratyphosum A* (*H*), and to *Bact. typhosum* (*O* and *H*). Reactions to *Br. abortus* and to a strain of leptospira were negative. There were strongly positive Wassermann and Sachs-Georgi reactions, these became almost completely negative after three months without anti-syphilitic treatment.

Guineapig inoculations were negative, both for the blood of the patient and for the brains of six rats from the ship.

Valid reasons are given for regarding the reactions as being compatible with the diagnosis of a typhus-like fever belonging to the tick-borne group. A fever of this kind is known to occur in São Paulo in South America and the other clinical features of the case favoured the diagnosis. The authors conclude by suggesting that "The patient was possibly infected by one or more ticks which had travelled from South America, either among a cargo of hides or infesting one of the rats on the ship he had been unloading." *J. W. D. M.*

VAN DER SCHROEFF (J. P.). Een epidemie van mijtekoorts en tropical typhus in Atjeh en Onderhoorigheden. [**An Epidemic of Mite Fever and Tropical Typhus in Atjeh and Dependencies.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1941. May 20. Vol. 81. No. 20. pp. 1103–1122. With 5 charts. English summary.

Atjeh is situated at the extreme northern extremity of Sumatra and the epidemic in question comprised 29 cases of mite fever and 48 of tropical typhus. The epidemic broke out among the coolie labourers engaged in clearing jungle growth for the planting of oil palm. In this area there had originally been a coffee plantation which had to be freed not only from coffee bushes but from great overgrowth of alang grass. Mice, rats and the lalang bird, with their parasites—mites (larvae of *Trombicula deliensis*) and ticks (Amblyomma)—were abundant. The coolies received wounds on the legs in the course of clearing operations. Work was begun in June 1939 with 26 coolies and the first four cases of tropical typhus made their appearance in July. By September an average of 178 coolies were at work and in October the number had risen to 216. In the latter month there were 3 cases of mite fever and 14 of tropical typhus, while in November, although the coolie strength had fallen to 39, there were 16 cases of each disease. Prophylactic measures were taken: these consisted in smearing the bare legs with a mixture: cocoanut oil 18 litres; cajuput oil 800 cc. By February 1940, when there were as many as 201 coolies at work, there were only 4 cases of tropical typhus and 1 case of mite fever.

It may be noted that clinical symptoms of tropical scrub typhus and mite fever are much the same. The latter disease shows a "primary effect" and the former none. The difference is explained by most workers as due to the fact that the mite pierces the skin only superficially and that, with the deposition of the virus, necrosis and reaction occur. The tick, on the other hand, has a long proboscis which reaches down to the subcutaneous tissue and no necrosis is produced. In both

affections there was swelling of regional lymph nodes and sometimes a rash was observed. The serum reaction for both is an agglutination of the *Kingsbury* strain of *Proteus*, but not of *X19* (shop typhus reaction).

In this article the author contends that both infections are the same and that both may have a primary effect, whether of mite or tick bite, although this is not always evident. A case mortality rate of 3 to 4 per cent. may be expected. Sulphonamides in this epidemic did not influence the clinical course of the disease. *W. F. Harvey.*

LEIMENA (J.). Een geval van scrubtyphus (Tropical Typhus). [**A Case of Scrub Typhus.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1941. Feb. 18. Vol. 81. No. 7. pp. 339–341.

Scrub typhus is found in the Dutch East Indies, particularly in Sumatra, but shop typhus is more prevalent in Java. Since 1931, patients with shop typhus, giving the Weil-Felix reaction with *Proteus X19*, have regularly been seen at the Immanuel hospital; the author now describes the second case of scrub typhus seen there, in a man from Bandoeng who had never been out of Java. No rash was seen, nor was there any evidence of primary sore; the Widal reaction was negative but the Weil-Felix with *Proteus OXK* was positive to 1 in 400. The fact that there was no primary sore leads the author to the opinion that this case resembled scrub typhus rather than the Sumatran mite fever [but LEWTHWAITE & SAVOOR (this *Bulletin*, 1940, Vol. 37, p. 847) have come to the conclusion that this matter of primary sore is not valid in differentiation between these diseases, which are all identical with tsutsugamushi]. *C. W.*

MAY (A. J.). **Endemic Typhus in Papua.**—*Med. Jl. Australia.* 1941. Apr. 12. 28th Year. Vol. 1. No. 15. pp. 449–450.

The author describes two cases of fever in Europeans in Papua. One patient had been working on newly cleared scrub land; his rise of temperature lasted for two weeks, ending by lysis; there was a rash of rose coloured patches on the 6th day but no eschar could be found. His serum on the 12th day agglutinated *Proteus XK* at a titre of 1 in 160 but failed to agglutinate *Proteus X19* or *X2*. In the second case serum tests were not performed but the course of the disease was similar to that in the first patient. Histories of similar fevers are not uncommon in Papua and the author considers it probable that the disease has long existed there in the natives, though this is the first occasion on which it has been proved. *C. W.*

KAWAHIGASI (K.). Erfahrungen an der Tutugamusi-Krankheit, besonders ihrer sog. atypischen Fällen. [**Atypical Tsutsugamushi Fever.**]—*Taiwan Igakkai Zassi* (*Jl. Med. Assoc. Formosa*). 1941. Feb. Vol. 40. No. 2. [In Japanese pp. 355–367. [13 refs.] German summary p. 367.]

In the last 3 years 10 cases of tsutsugamushi fever have been observed and in 5 of these no primary sore could be discovered. It is therefore considered that atypical tsutsugamushi fever is common in Formosa. The only difference noted between the typical and atypical fevers is the presence of the primary sore in the former. *D. H.*

KOUWENAAR (W.) & ESSEVELD (H.). Onderzoekingen over Rickettsiosen ter Oostkust van Sumatra. XII. Onderzoek naar de beschermende werking van mijtekoortsreconvalescentenserum van mensch en cavia. [**Rickettsioses of Sumatra. XII. Protective Action of Mite Fever Convalescent Sera from Man and Guineapig.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1941. June 3. Vol. 81. No. 22. pp. 1203–1217. [26 refs.] English summary.

The degree of immunity developed after mite fever has not yet been settled. It is not uncommon for persons to have two attacks of the disease, and immunity to intratesticular injection in monkeys and rabbits is only established in a certain proportion of the cases. A difficulty arises in experiments with guineapigs owing to the fact that the great majority of test animals do not show fever at all. Strains were found, however, especially the Seerangayee strain, which did give a fever reaction in some 80 per cent. of the guineapigs. It was now possible to commence immunity experimentation and the authors have concentrated their attention on such characters as (1) the duration of the incubation period or the fever in dead and recovered guineapigs, (2) the protective action of human and guineapig convalescent serum, and (3) the viricidal action of convalescent serum.

Their results are summarized as follows :—(1) Guineapigs after a mite fever infection remain immune to a second injection for at least 13 months. (2) Convalescent serum from either guineapig or man fails to protect a guineapig against an infection with the virus whether the serum be administered before, along with, or after the virus. No beneficial effect on the gravity of the illness was apparent from the use of convalescent serum. In fact, the serum showed no indication of any therapeutic effect. (3) Even if serum and virus were mixed and left in contact for two hours before injection the course of the infection could only be slightly attenuated. [Compare the results with experimental Rocky Mountain Fever, this *Bulletin*, 1940, Vol. 37, p. 575.]

W. F. Harvey.

DURAND (Paul) & GIROUD (Paul). Le lapin inoculé par voie respiratoire avec les rickettsies du typhus historique. Pouvoir antigène des suspensions. [**Rabbits Inoculated by the Respiratory Route with Rickettsiae of Epidemic Typhus. The Antigenic Power of Suspensions.**]—*Ann. Inst. Pasteur.* 1941. June. Vol. 66. No. 6. pp. 425–437.

A detailed account is given of the preparation of vaccines against epidemic typhus by inoculating rabbits with *Rickettsia prowazeki* by the tracheal route. As rabbits are less susceptible than mice to lung infection, their resistance had to be lowered by such means as the injection of toxic antigens, shaving the skin, or exposure to low temperatures.

Each rabbit was given a suspension made from the consolidated lung of a mouse which had received tracheal injections of Rickettsiae from infected lice about three days previously. The rabbits developed a solid pneumonia with red or grey hepatization within five to seven days. In some cases smears from the lungs showed abundant Rickettsiae.

Vaccines were prepared from the lungs in the same way as had already been adopted by the authors in the case of mice. Although

the antigenic value of the suspensions was lower than that of mouse vaccines it was sometimes possible to prepare more than thirty times as much vaccine from a rabbit as from a mouse, owing to the greater size of the animal's lung.

When the lungs of the rabbits contained few or no visible Rickettsiae the antigenic power of suspensions made from them was insignificant.

The immunizing power of vaccines made from lungs rich in Rickettsiae was found to be equal to that of mouse vaccines, as shown by protection tests on guincapigs and the Weil-Felix response in human beings.

For details of the methods employed and the pitfalls encountered reference must be made to the original paper.

The experimental work described in the paper was carried out at the Pasteur Institute, Paris, between November 1939 and April 1940, but, for reasons which can readily be surmised, it could not be published till July 1941. *J. W. D. M.*

FINLAYSON (M. H.) & GROBLER (G. M.). **Vaccination against Epidemic Typhus in South Africa.**—*South African Med. Jl.* 1941. Apr. 12. Vol. 15. No. 7. pp. 125–127.

In this research the authors utilized the Zinsser-Castaneda (1931) vaccine subcutaneously in guineapigs and subsequently tested the animals with a varying number of infective doses of South African epidemic typhus virus.

It was noted that 3 doses of 1 cc. of the vaccine conferred complete protection on 22 of 24 animals tested, but that 3 doses of 0·25 cc. protected all of 6 tested, whereas a single dose of 1 cc. of vaccine only protected 50 per cent. of the animals tested.

It was found also that this immunity lasted for at least a year after the inoculations. The great difficulty in inoculating large numbers of African natives lies in administering two or more doses of vaccine, but it has been shown that alum-treated vaccines will protect 80 per cent. of animals when given in a single dose of 0·5 cc. *D. H.*

LANCET. 1941. Sept. 6. p. 289. [12 refs.]—**Relationship of Epidemic and Endemic Typhus.**

In this editorial there is a useful résumé of the work recently done on the immunization of animals against epidemic typhus strains by means of vaccines prepared from murine strains. *C. W.*

RUIZ-CASTANEDA (M.). **Active Immunization against Epidemic Typhus by means of Vaccines prepared from Endemic Virus.**—*Brit. Jl. Experim. Path.* 1941. June. Vol. 22. No. 3. pp. 167–172. [12 refs.]

This article describes a practical method of preparing large quantities of vaccine from the lungs of rats infected by intranasal instillation of Rickettsiae of the murine type.

The vaccine protected guineapigs against infection by Rickettsiae of the classical strain of European typhus, but larger doses were needed than those which were effective against murine virus. The author writes that " it is impossible to state from the information at hand whether or not there are essential antigenic differences between

the endemic (murine) and epidemic viruses," but he concludes that such differences are not enough to justify disregard of vaccines prepared from endemic strains as practical immunizing agents against classical typhus virus. *J. W. D. M.*

PARKER (R. R.). **Rocky Mountain Spotted Fever : Results of Fifteen Years' Prophylactic Vaccination.**—*Amer. Jl. Trop. Med.* 1941. May. Vol. 21. No. 3. pp. 369–383. With 2 charts.

In this article Parker deals with the protective value of killed vaccine made from the tissues of infected ticks (*Dermacentor andersoni*).

In the western Montana area, where the case-fatality rate is very high, 95 attacks occurred during 15 years ; of 51 non-vaccinated cases 42 were fatal (82·35 per cent.), of 37 cases in which the attack occurred in the same year as the last vaccination only 3 died (8·11 per cent.) and the average severity of the attacks was greatly reduced. The remaining 7 cases were left out of account because protection is claimed only for the year in which vaccination is carried out and they had been vaccinated from 1 to 5 years before the attack.

The vaccine, therefore, gives a high degree of protection against death but little protection against attack in areas of highly virulent infection.

In South Idaho, where the case fatality is low (about 5 per cent.), there were 22 cases in a control group of 364 non-vaccinated persons during the period of observation in 1926–27, and only one case among 193 vaccinated persons.

In areas of infection of low virulence vaccination gives almost complete protection against attack.

Apart from these test areas 455,000 persons were vaccinated and only 29 of these were attacked within a year of being vaccinated ; there were no deaths.

It was also found that persons vaccinated in two or more successive years were much better protected than those who were only once vaccinated. A dose of the vaccine given within a day or two after the bite by an infected tick modified the severity of the attack in areas where mild infections predominated and the incubation period was usually prolonged.

The only condition in which vaccination is not recommended is when a bite by a suspected tick has already occurred in areas of highly virulent infection. *J. W. D. M.*

BURNET (F. M.) & FREEMAN (Mavis). **Studies of the X Strain (Dyer) of *Rickettsia burneti*. II. Guinea Pig Infections, with Special Reference to Immunological Phenomena.**—*Jl. Immunology.* 1941. Apr. Vol. 40. No. 4. pp. 421–436. With 4 figs.

This research was carried out in Australia with a strain of virus, sent from America, which had been isolated from a patient infected in the laboratory in Montana. It was confirmed by the authors that this X virus is considerably more virulent for guineapigs than the local strain of virus. Fatal infection frequently resulted and fibrinous exudate was noted in the spleen. Numerous Rickettsiae were found in this exudate and in the tissues. It was also confirmed that the X strain (American) and the local strain of virus from cases of Q fever are immunologically identical. In guineapigs large doses of X virus

produced fever in 24 hours and this fever continued till death. With medium doses a primary rise was noted, followed later by a period of fever lasting 6 to 18 days.

When the virus was previously mixed with immune serum and then injected no fever resulted, but when tested later the animals were found to be immune ; similar results were obtained when the virus was treated with lauryl sulphate solutions.

Rickettsiae killed or inactivated by formalin or by heat produce a substantial immunity in guineapigs. *D. H.*

BURNET (F. M.) & FREEMAN (Mavis). **Studies of the X Strain (Dyer) of** *Rickettsia burneti.* **I. Chorioallantoic Membrane Infections.**—*Jl. Immunology.* 1941. Apr. Vol. 40. No. 4. pp. 405–419. With 3 figs. [19 refs.]

Employing the X strain (American) of Q fever virus the authors carried out a series of experiments on the inoculation of egg membranes by the modified Goodpasture technique. Younger embryos 7 to 9 day old were readily infected ; in the older 12-day embryos there was some evidence of resistance and patches or foci of infection could be seen and counted. The histological changes in these areas were similar to those produced in egg culture by other virus diseases ; these changes were limited to the superficial layer of cells and showed oedematous and vacuolated cells containing Rickettsiae. None of the immune sera at hand showed any definite activity in preventing the formation of specific foci in the egg membrane or reducing the number of those on the older embryos. It was noted that the virulence of the infection for guineapigs was much increased after culture in the egg membrane and this increase of virulence is compared with that noted in the tick with the virus of Rocky Mountain fever ; the explanation given for this increased virulence is that in both instances no antibody is formed, whereas in infected animals antibody is produced.

D. H.

DAVIS (Gordon E.). *Rickettsia diaporica:* **its Persistence in the Tissues of** *Ornithodoros turicata.—Public Health Rep.* 1940. Oct. 11. Vol. 55. No. 41. pp. 1862–1864.

Thirty-eight *O. turicata* were fed on a guineapig ill with the original Montana strain of American Q fever. They were subsequently tested at irregular intervals for transmission of *R. diaporica* and for the presence of this organism in their tissues. In the first test they were allowed to engorge on and detach voluntarily from guineapigs, and in no case was the disease transmitted during this process. In the second test the ticks were ground up and injected ; by this test it was shown that *R. diaporica* may persist in the ticks for 1,001 days after the infective feed. Tests to determine if the ticks could transmit the infection to their progeny were all negative. It was shown that the excreta of the ticks were infective on injection into guineapigs. The authors recall that *O. turicata* may sustain *Bact. tularense* in its tissues for 674 days, does not transmit that organism while feeding or to its progeny, and that, as also with *R. diaporica,* the virulence is not affected by long residence in the tick or by prolonged fasting of the tick. *C. W.*

SMITH (D. J. W.). **Studies in the Epidemiology of Q Fever. 8. The Transmission of Q Fever by the Tick** *Rhipicephalus sanguineus.*—*Australian Jl. Experim. Biol. & Med. Sci.* 1941. June. Vol. 19. Pt. 2. pp. 133–136.

Experiments carried out at Brisbane and described in this important paper demonstrate that *Rhipicephalus sanguineus* must be regarded as a potential vector of Q fever. Hundreds of ticks and about 60 guinea-pigs were used. Larval, nymphal and adult ticks were infected with Q fever by feeding them on infected guineapigs.

The virus was shown to persist in nymphs which had been infected in the larval stage and in adults infected as nymphs, but infection was not transmitted from infected adults to their progeny.

Infected nymphs transmitted infection to 63 per cent. of the guinea-pigs on which they fed: in the case of adults the infection rate was 92 per cent.

Rickettsiae were seen in 29 out of 40 smears made from adult ticks which had previously been exposed to infection in the larval or nymphal stage and in 11 of 23 cases in which serial sections were made of adult ticks belonging to the same batch. Faeces collected from infected adult ticks were found to be highly infective to guineapigs: 65 days after collection the infecting dose was one-hundred-millionth of a gramme. *J. W. D. M.*

SMITH (D. J. W.). **Studies in the Epidemiology of Q Fever. 7. The Biology of** *Haemaphysalis humerosa* **Warburton and Nuttall (Acarina, Ixodidae) in Queensland.**—*Australian Jl. Experim. Biol. & Med. Sci.* 1941. Mar. Vol. 19. Pt. 1. pp. 73–75. With 1 fig.

BENGTSON (Ida A.). **Studies on Active and Passive Immunity in "Q" Fever Infected and Immunized Guinea Pigs.**—*Public Health Rep.* 1941. Feb. 21. Vol. 56. No. 8. pp. 327–345. With 9 figs.

Rickettsial active and passive immunity in experimental animals as shown by the Q fever virus follows in general the same laws as those which apply in bacterial active and passive immunity. Active immunity results when the disease has run its course in the animal or when killed organisms are introduced into the body. Evidence of immunity is found in resistance to reinfection with the disease and in the development of agglutinating and neutralizing antibodies. Sera from animals which have recovered from the disease or which have received killed cultures confer passive immunity when mixtures of the immune serum and the infecting agent are inoculated into animals. Active immunity can be readily produced in experimental animals by the use of killed Rickettsial vaccines. Hyperimmune sera may be produced in guinea-pigs and in rabbits by the inoculation of killed cultures followed by the injection of living organisms; the titre of agglutination in the rabbit sera, however, in no instance rose above 1 in 640. *D. H.*

BENGTSON (Ida A.). **Complement Fixation in "Q" Fever.**—*Proc. Soc. Experim. Biol. & Med.* 1941. Apr. Vol. 46. No. 4. pp. 665–668. [11 refs.]

In this paper a complement fixation reaction for Q fever, which is specific among the Rickettsial diseases, is described. Yolk sac antigen was found to be superior to mouse spleen antigen in tests when human

sera were used. The test was shown to have a good degree of sensitivity, it appeared in 9 to 13 days after the onset of the disease and persisted for at least 305 days. The indications are that the test is of value for diagnosis and that it affords evidence of immunity. *D. H.*

HORNIBROOK (J. W.); NELSON (K. R.); DYER (R. E.); TOPPING (N. H.); BENGTSON (I. A.). **An Institutional Outbreak of Pneumonitis. I. Epidemiological and Clinical Studies** [HORNIBROOK & NELSON].—*Public Health Rep.* 1940. Oct. 25. Vol. 55. No. 43. pp. 1936–1944. With 1 fig. & plates 1–4. **II. Isolation and Identification of Causative Agent** [DYER, TOPPING & BENGTSON].—*Ibid.* pp. 1945–1954. With 5 figs. (3 on plates 5–6).

This report describes in detail a remarkable outbreak of infection by the Rickettsiae of Q fever. Between the end of March and the middle of May 1940, 15 cases of fever with one death occurred among 153 employees in one building of the National Institute of Health at Washington where strains of Australian and American Rickettsiae of Q fever had been under investigation since 1938.

The fever lasted from 5 to 15 days except for one slight case in which the duration was only 2 days.

The most remarkable feature of the disease was that although there were practically no physical signs, all the patients showed X-ray evidence of a patchy central pneumonia.

Blood counts and cultures gave negative results; so did agglutination tests against *Bact. typhosum, Bact. paratyphosum A* and *B, Brucella abortus, Proteus OX19* and *Bact. tularense.*

The causative agent was identified by Dyer, Topping, and Bengtson as the filter-passing Rickettsia of Q fever [also known as *R. burneti* and *R. diaporica*].

This organism was isolated from three out of four patients in whose cases attempts were made to find the virus: it proved to be identical in all respects with the strains which were under investigation in the building, but a curious feature of the outbreak was that no cases occurred in the wing in which the Rickettsiae were being studied.

The source of infection was not discovered; the inhalation of infected dust raised by sweeping the floors seemed to be the only hypothesis which was supported by the evidence.

Reference is made to eight recent reports of outbreaks of a similar type of disease variously designated as "acute pneumonitis," "atypical pneumonia," "atypical broncho-pneumonia," "virus pneumonia," etc. All but one of these reports have been from the U.S.A. The authors state that it is a matter of conjecture whether these were caused by Rickettsiae.

[The high degree of susceptibility of mice and other animals to severe pneumonia resulting from the introduction of the Rickettsiae of the various fevers of the typhus group seems to favour the tentative suggestion by the authors that this outbreak may have been caused by dust-borne infection.] *J. W. D. M.*

HESDORFFER (M. B.) & DUFFALO (J. A.). **American Q Fever. Report of a Probable Case.**—*Jl. Amer. Med. Assoc.* 1941. Apr. 26. Vol. 116. No. 17. pp. 1901–1902. With 2 figs. [16 refs.]

The patient contracted a fever while working in the woods in Montana; although there were no signs of pulmonary disease, an

X-ray of the chest was taken and revealed patchy pneumonitis. There was abdominal discomfort. Treatment with sulphathiazole was given and the temperature came down by lysis to normal on the eighth day. The Weil-Felix test with *Proteus X19* was negative on the fifth day, as were agglutination tests for the typhoid group, *Br. abortus* and *Bact. tularense*, but on the seventh day *Rickettsia diaporica* was agglutinated at titre 1 : 80, and three weeks later at 1 : 160. The patient's blood was not infective for guineapigs on the seventh day. There was no history of tick bite, but on the evidence of the agglutination test and the central pneumonia the case is regarded as probably one of Q fever ; this disease is probably more widespread than is at present known. *C. W.*

WALKER (Allan S.) & DODS (Lorimer). **Clinical Impressions of an Epidemic of Sandfly Fever in Palestine during 1940.**—*Med. Jl. Australia.* 1941. Mar. 22. 28th Year. Vol. 1. No. 12. pp. 345–349. With 5 figs.

This paper does much more than is suggested by the modest title, " clinical impressions " ; it gives a remarkably vivid description of the clinical features of an epidemic of sandfly fever in which 1,500 patients were seen at the First Australian General Hospital in Palestine from May to November 1940. No fatalities are mentioned, presumably none occurred.

Although sandfly fever is negligible as a cause of permanent casualties it has a real military importance, so that a useful purpose would be served by circulating reprints of this excellent description of the disease to all Medical Officers who are serving, or are likely to serve, in countries where outbreaks are liable to occur.

Only a few points can be referred to in this review.

The fever lasted more than three days in 90 per cent. of the cases so that the name " three days fever," sometimes applied to the disease, is obviously inappropriate.

Secondary rises of temperature occurred in " a number of cases " ; sometimes the fever recurred after several days and was accompanied by a recrudescence of the symptoms.

Later recurrences were unusual but a second attack was observed within several weeks or months in some instances. This observation corrects the impression conveyed by some accounts of the disease that a solid and lasting immunity is conferred by one attack.

A sense of band-like constriction round the lower part of the thorax was " so curiously prominent in a number of cases that it was reminiscent of the epidemic pleurodynia known as Bornholm disease." [This feature of the epidemic supports a suspicion long entertained, but not hitherto expressed, by the reviewer that Bornholm disease may be a virus disease belonging to the dengue-sandfly-fever group of diseases and conveyed by an insect, possibly a midge.]

In the remarks on differential diagnosis the authors state that " the more severe nature of mosquito dengue fever may be pointed out " ; but in many epidemics of dengue the symptoms are not more severe than those described in this paper ; in addition to the greater frequency of skin rashes mentioned by the authors, the greater frequency of secondary rises of temperature in dengue is a more striking point of difference than the intensity of the symptoms.

An almost constant feature was the presence of a crop of vesicles on the palate : an identical eruption on the palate was found in cases of infective hepatitis (infectious non-leptospiral jaundice) which occurred in a number of men during the period of the epidemic of sandfly fever. In both diseases there were also tenderness on pressure, and pain on movement, of the eyeballs with variable conjunctival congestion, together with an absence of herpes febrilis.

The authors point out that these features have been observed in several virus diseases and lend support to the view that infective hepatitis is a virus disease. *J. W. D. M.*

PEARSON (A. S.). **Sand-Fly Fever and Benign Lymphocytic Meningitis.** —*Brit. Med. Jl.* 1941. Aug. 30. pp. 303–304.

This report deals with 8 cases of benign lymphocytic meningitis and 7 of sandfly fever which occurred in a "general military hospital" between August 7th and December 12th, 1940.

The clinical features of the two groups of cases were remarkably similar so that several cases which were thought to be lymphocytic meningitis proved to be sandfly fever and *vice versa*.

The following extract from the tabulated statement of symptoms and physical signs shows that apart from the pronounced increase in the lymphocytes of the cerebrospinal fluid there was no single feature by which every case of lymphocytic meningitis could be distinguished from sandfly fever :—

	Benign Lymphocytic Meningitis (8 cases)	Sandfly Fever (7 cases)
Orbital pain	8	5
Photophobia	4	2
Frontal headache	8	7
Occipital pain	5	2
Vomiting	6	1
Pain in neck	7	5
Pain in muscles	1	4
Pain in back	4	2
Neck rigidity	7	2
Absence of biceps reflex ...	5	6
,, supinator reflex	4	6
Positive Kernig	4 (2 slight)	1 (left side)

The average temperature and pulse rate were higher in the cases of sandfly fever. The lymphocytes in the cerebrospinal fluid ranged from 72 to 332 per cmm. in the cases of meningitis against 1 to 5 in those of sandfly fever.

It must be emphasized that the cases of sandfly fever were not average and unselected, they were only those in which certain features suggested a possible diagnosis of lymphocytic meningitis so that a lumbar puncture was justifiable.

Major E. H. EVANS, R.A.M.C., who is expected to make a further report on the subject, pointed out to the author that the curves of seasonal incidence of the two diseases were almost identical. One case of the meningitic type occurred 24 days after the end of the "official" season for sandfly fever, but a sandfly was found a few days after the admission of the patient.

The author concludes—" It would therefore appear that benign lymphocytic meningitis is made up of a group of diseases, and that the virus of sand-fly fever is capable of causing the condition."

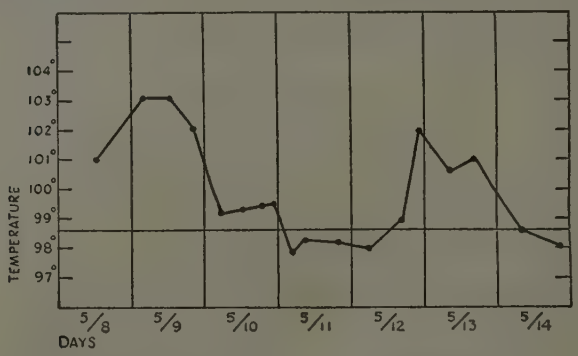
[As this form of meningitis sometimes occurs in a number of other diseases, such as mumps, measles and anterior poliomyelitis, it might preferably be regarded as an occasional feature of these infections rather than as a " group of diseases."] *J. W. D. M.*

TOPPING (Norman H.), CULLYFORD (James S.) & DAVIS (Gordon E.). **Colorado Tick Fever.**—*Public Health Rep.* 1940. Nov. 29. Vol. 55. No. 48. pp. 2224–2237. With 5 figs.

This interesting report deals chiefly with 11 cases of Colorado tick fever which occurred in May and June, 1940, and were closely investigated by members of the U.S.A. Public Health Service.

All the cases showed a close clinical resemblance to dengue in every detail except that there was no rash.

The fever always came in two spells of 1 to 3 days' duration separated by a fever-free interval of 1½ to 3 days : the total duration of the fever was never more than 7 days.



Typical temperature record in a case of Colorado tick fever.
[Reproduced from *Public Health Reports.*]

There was pronounced leucopenia ranging from 1,300 to 4,500 leucocytes per cmm. Blood cultures, animal inoculations and agglutination tests were negative in all the cases.

The epidemiology was quite different from that of dengue ; there was no evidence of person to person infectivity and the vector was a tick, *Dermacentor andersoni.*

All the patients had been recently bitten by a tick, usually 4 or 5 days before the onset. All the cases occurred during the period of activity of the vector tick.

Information was collected from physicians who had reported 53 cases in the locality during 1940 : most of these were similar to the 11 cases described by the authors but some of them had only one spell of fever and in some the duration was more than 7 days. The authors state that " it is obvious that some of the cases were not Colorado tick

fever as we know it" : they also point out that the popular name of the disease is misleading because it undoubtedly occurs in some of the surrounding States.

[The evidence strongly favours the view that the disease is caused by a virus closely related to that of dengue and transmitted by a tick, presumably from an animal of the wilds. The disease may be provisionally classified as a non-epidemic fever belonging to the dengue group and the name "tick dengue" would be descriptive of its chief features.] *J. W. D. M.*

PATIÑO-CAMARGO (Luis). El primer caso de bartonellosis (fiebre verrucosa del Guáitara o Verruga) en Bogota. [**The First Case of Guáitara Fever in Bogotá.**]—Reprinted from *Rev. Facul. de Med.* Bogotá. 1940. Dec. Vol. 9. No. 6. 11 pp. With 5 figs & 1 chart. [13 refs.] English summary.

A cavalry corporal, 24 years of age, was admitted to hospital with symptoms which were at first diagnosed as due to typhoid fever [the symptoms as detailed—a little anaemia, meteorism, palpable spleen, rapid heart action—seem hardly to warrant such a diagnosis without further confirmation and blood examination]. He died 40 days later. Seventeen days before his death blood was taken, as bartonellosis was then suspected, and growth was obtained in Noguchi's medium in 10 days and the organism confirmed by inoculation of monkeys, a *Macacus rhesus* and a *Cebus fatuellus*.

[It is probably true that this is the first case of this condition *observed* in Bogotá, but, as is acknowledged in the text, the patient had a few months before been in Nariño, where Guáitara fever is common. "During the year 1939, 1448 patients died from verruga in the Department of Nariño" among a population of about 150,000. (See also this *Bulletin*, 1940, Vol. 37, pp. 271, 582, 583.)] *H. H. S.*

GEIMAN (Quentin M.). **New Media for the Growth of** *Bartonella bacilliformis*.—*Proc. Soc. Experim. Biol. & Med.* 1941. June. Vol. 47. No. 2. pp. 329–332. [Summary appears also in *Bulletin of Hygiene*.]

Media liquid, semi-solid and solid; tissues and developing chick-embryos have all been used for growing *Bartonella bacilliformis*; Noguchi's semi-solid medium for leptospira and nutrient blood agar have proved the most serviceable but even on these growth has been slow and relatively sparse. The observation had been made that in leptospira medium there was need of serum and haemoglobin from laked corpuscles, indicating the need for "certain vitamins and growth-promoting factors found in the cellular elements of the blood." Also, in disease the organism multiplies within the reticulo-endothelial cells "suggesting the possible advantageous use of ingredients in monocyte media." The author has used for his experiments five strains from Peru, from cases of Oroya fever, verruga peruana, and the proboscis of a sandfly, *Phlebotomus verrucarum*, and finds the following media quite satisfactory.

1. *Liquid tryptone-serum*, prepared as follows :—

"The liquid tryptone-serum medium is mixed as follows : 75 cc. of a sterile 1% solution of tryptone in distilled water adjusted to pH 7·6–7·8, 25 cc. of fresh rabbit serum, and 0·2 cc. of a sterile (Berkefeld N) mixture

of ascorbic acid-glutathione solution (10 mg. ascorbic acid and 40 mg. glutathione in 20 cc. Ringer's solution). Place 8 or 10 cc. of this mixture in small sterile Erlenmeyer flasks (50 cc.), incubate for sterility and then inoculate with bartonella. Growth (maximum at 28°C.) takes place on the bottom of the flask, becoming visible as a finely divided sediment within 24 to 48 hours and reaching a peak on about the tenth day. Depending upon the size of the original inoculum, an amount up to 0·1 cc. of packed organisms can be harvested from each flask. These organisms can be resuspended in 3–5 cc. of buffered saline solution pH 7·8–8·0 to make a homogeneous suspension for use in testing immune sera for agglutinins.

2. *Semi-Solid Medium.*

" The semi-solid medium is made as follows: Base agar—2 g. Bactoagar, 8·5 g. sodium chloride, 1000 cc. distilled water. Dissolve ingredients and adjust to pH 7·6–7·8. To 100 cc. of this base medium, dissolved and cooled to 45°C., add 10 cc. of sterile 5% aqueous tryptone adjusted to pH 7·6–7·8, 10 cc. of fresh sterile rabbit or sheep serum, and 0·2 cc. ascorbic acid-glutathione solution. Dispense in sterile test tubes, incubate, insert rubber stoppers to prevent evaporation if desired, and inoculate. At 28°C., growth becomes grossly visible at 36 to 48 hours and very heavy at about the fifth day."

The medium is transparent and colourless, so that detection of growth is easy. At room temperature growth is slower and subcultures may be made every 6–8 weeks.

3. *Solid Medium.*

" The solid medium is made as follows: Base agar—20 g. Bactoagar, 20 g. tryptone or proteose peptone No. 3, 5 g. sodium chloride dissolved in 1000 cc. distilled water and adjusted to pH 7·6–7·8. To 75 cc. of sterile and dissolved base agar cooled to 45°C., add 25 cc. of fresh (rabbit or sheep) serum or, for economical reasons, 25 cc. of whole defibrinated blood from rabbits or sheep, and 0·2 cc. ascorbic acid-glutathione solution. Place sufficient quantities in test tubes to make a short butt and a long slant. When slants are set, place sterile rubber stoppers in the tubes and incubate for sterility. Inoculate the slant with *B. bacilliformis* by the loop method and incubate at 28°C. Growth follows the streak of the loop, becoming grossly visible within 24 to 48 hours and reaching a maximum in 10 to 14 days. Heavy growth takes place also in the water of condensation. The transparency of the medium containing serum facilitates observation of growth and general morphology of colonies."

Defibrinated blood may be used in place of serum, and there is then no need for including blood cells or laked blood.

Growth with any of these media is much more luxuriant than with the leptospira or blood agar medium, permitting immunological and other investigations which may lead to measures of control of Carrión's disease. *H. H. S.*

Ristorcelli (A.) & Dao van Ty. Phlébotomes d'une région de Colombie où la verruga du Pérou est devenue endémique depuis trois ou quatre ans. [**Sandflies of an Endemic Area of Verruga Peruviana in Colombia.**] —*Ann. Parasit. Humaine et Comparée.* 1941. Vol. 18. No. 1–2–3. pp. 72–74.

LEPROSY.

Précis of Abstracts in this Section.

Manalang (p. 698) discusses the seasonal incidence of leprosy in the Philippines, with special reference to relapses. Malhotra (p. 698) refers to the unusual distribution of leprosy in the Kangra district of the Punjab, where the lepromatous form is as common as the neural. Chandy (p. 698) reports a higher rate in high-caste Brahmins than in those of lower castes in the Fyzabad district in India.

Motta and Costa (p. 699) give information on leprosy in the Federal District, Rio de Janeiro, and conclude that the disease is spreading rapidly in that area. Fonte (p. 699) reports on leprosy in Hawaii.

Chaussinand (p. 699) gives an account of attempts, carried out over a period of seven years, to cultivate *Myco. leprae*. Loving (p. 700) reports success in attempts to infect rabbits with the Duval acid-fast bacillus; the resulting lesions were microscopically identical with those of human leprosy.

Lowe and Chatterji (p. 700) show that more than half of the lepromatous cases investigated commenced with symptoms of the neural type. The neural phase usually lasted 3–4 years, and long-standing neural cases rarely became lepromatous. They (p. 701) record a case of leprosy in a child in which the disease developed from a few depigmented patches to diffuse lepromatous infiltration. The same authors (p. 701) report an abortive case of neural leprosy, and (p. 701) describe the acute onset of lepromatous disease in a man aged 70. Velasco (p. 702) reports two cases in which tuberculoid disease developed into a lepromatous form.

From experiments with several acid-fast bacilli which at some time have been suspected of causing leprosy, Dharmendra and Bose (p. 702) conclude that complement fixation tests with sera from lepers do not give any evidence regarding the part played by these bacilli in causing the disease. Eagle *et al.* (p. 703) show that in leprosy, when syphilis is not present, the complement fixation test, in which a suspension of *Spirochaeta pallida* is used, is negative, although when mammalian tissue antigen is employed it may be positive.

From a study of the lepromin test in children in Madras, Cochrane *et al.* (p. 703) conclude that the reaction tends to be negative in those in whom a history of contact is maximal, but is not significantly influenced by hereditary predisposition. Wade (p. 703) confirms the previously reported fact that the normal dog reacts positively to intracutaneous injections of lepromin, and is capable of developing an allergic state to the antigen. In human leprosy of the lepromatous type the specific feature is the loss of this power.

Collier (p. 704) in Thailand reports favourably on the use of diphtheria toxoid in treatment. Moiser (p. 704), however, found it to be ineffective in a series of cases in Southern Rhodesia. Davison (p. 705) failed to obtain any useful results in treatment with blood transfusions. Kuznezov (p. 705) reports favourably on the use of medicinal baths of naphthalan oil in Russia.

Baliña and Basombrio (p. 705) record favourable results in a series of cases in which primary lesions of a tuberculoid nature were removed or destroyed by cauterization.

MUIR (p. 706) reports high incidence of lepromatous ulcers in Trinidad, and outlines a treatment with gentian violet, silver nitrate and tannic acid, which has given good results. SLOAN (p. 706) has had success in the treatment of perforating ulcers with urea.

Reviewing the reports of various conferences SEN (p. 706) emphasizes that control can only be obtained by isolation of patients from young people, but that it may be aided by treatment and education. DAVEY (p. 706) reports on the progress of a series of untreated cases during two years at a village in Nigeria where isolation was enforced by the villagers themselves, the patients living in a small leper village. There was evidence of improvement in the position.

ROGERS (p. 707) discusses the voluntary sterilization of married male lepers. *C. W.*

MANALANG (J.). **Influence of Climatic Changes on "Interruptions" and "Relapses" in Leprosy.**—*Jl. Philippine Med. Assoc.* 1941. Mar. Vol. 21. No. 3. pp. 121–126. With 1 fig.

The author has studied the seasonal incidence of leprosy in the Philippines, in relation to climatic conditions, on very similar lines to the work of LOWE and CHATTERJI in Calcutta [see this *Bulletin*, 1939, Vol. 36, p. 1015]. He classes the changes in his cases as "interruptions" when lepra bacilli reappear in the tissues and "relapse" when clinical signs also recur. "Interruptions" were slightly more numerous in the cool period, as were "relapses" to a still less degree, but he thinks the seasonal differences too slight to be other than accidental in nature, for the temperature variations ranged over only 5° centigrade against 13° in Calcutta. *L. Rogers.*

LEPROSY IN INDIA. 1941. Apr. Vol. 13. No. 2. pp. 48–49.—**A Note on Leprosy in the Kangra District of the Punjab.**

This note was prepared from information supplied by Dr. MALHOTRA, District Leprosy Officer, Kangra District. The district has more leprosy than the rest of the Punjab, and the known incidence is 0·1 per cent. of the population. The incidence is highest at 4,000 to 6,000 feet above sea level, and it is noted that the proportion of lepromatous cases is 54 per cent. of the whole at levels above 2,000 feet. This is the first area in India in which the number of lepromatous cases equals that of neural cases; as a rule the proportion is one to three or more. Of the known cases, 8 per cent. were 15 years of age or less; 56 per cent. were over 35. This is a rather surprising distribution in an area in which there are so many lepromatous cases. Tuberculoid disease is rarely seen.

The epidemiology of the disease, therefore, shows considerable differences in Kangra from that in most parts of India. *C. W.*

CHANDY (P. J.). **Leprosy in the Fyzabad District.**—*Leprosy in India.* 1941. Jan. Vol. 13. No. 1. pp. 18–22.

This report, based on 1,000 cases seen in two and a half years at a hospital in India, is mainly of local interest. A classification according to caste revealed a considerably higher rate among the high caste Brahmins than among the lowest castes. Two-thirds of the cases were neural and one-third lepromatous. *L. R.*

MOTTA (Joaquim) & COSTA (Henrique de Moura). A situação da lepra no Distrito Federal. [**Leprosy in the Federal District, Rio de Janeiro.**]—*Folha Med.* 1941. May 25. Vol. 22. No. 10. pp. 112–115.

This study was carried out in three and a half months, November 1940 to February 1941. First, from inquiries the authors collected information in 1,206 cases of leprosy, of whom 561 were living at home or in hospital (46·5 per cent.), 58 (4·8) had been transferred from other States, 187 (15·5) had died and 397 (32·9) could not now be traced. [These total 1,203 only, and the percentages, as given by the authors, are wrongly calculated.] They then made a study to determine the actual prevalence at the time and reach a total of 1,507, of whom 871 (57·7 per cent.) lived at home and 636 (42·3) were in institutions; 522 at Curupaiti and 114 in a leper hospital. Of the total, 1,428 (94·7) were adults and 79 (5·2) were children, and of the adults 801 (56·0) were men and 627 (43·9) were women. One thousand and forty-three were white, 185 coloured, 127 black, while of 152 the race was not known. As regards occupation those in domestic service were most numerous, 244, next, but far below, were artisans (73) and labourers (33). Of 1,049 indigenous patients 562 were from the Federal District and 487 from other States, 53·5 and 46·4 per cent. respectively. Of the total 1,507, 976 (64·7) were in the infective stage and 531 (35·2) burnt out or non-infective.

In their conclusions the authors state that the disease is extending rapidly in the Federal District, having increased by 50 per cent. in the past decade, that almost two-thirds are in the infective stages and that there is an "alarming deficit in the possibilities of internment."

H. H. S.

FONTE (Joir). A lepra no Hawaii. Quinze anos de atividade do centro de leprologia de Honolulu (Hawaii)—(1925–1939). [**Leprosy in Hawaii. Fifteen Years' Work of the Honolulu Centre.**]—*Acta Med.* Rio de Janeiro. 1941. May. Vol. 7. No. 5. pp. 236–244. English summary (8 lines).

After discussion by the Health Authorities of Hawaii there was inaugurated in November 1865 the Kalihi Hospital and a Detention Station for lepers in the suburb of Honolulu. In 1905 a leprosy Investigation Station was opened and two years later a crèche was founded in Molokai; in 1908 a Kahili Boys' Home was established for children of lepers and in 1909 the leprosarium of Kalawau.

This paper gives brief notes of statistical returns and clinical forms of cases seen in the fifteen years 1925–39; the former for 1925 and 1927, which have little, if any, interest now, the latter for the earlier years of the fourth decade of the century. Most of the figures refer to conditions of ten years or more ago and are not worth quoting here. Future papers will deal with laboratory, epidemiological, and bacteriological studies, the treatment of the disease and with murine leprosy.

H. H. S.

CHAUSSINAND (R.). Contributions à l'étude de la lèpre. I. Essais de culture du bacille de Hansen. [**Attempts to cultivate** *Myco. leprae.*]—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 69–75.

This is an account of a laborious seven years' investigation of numerous culture media inoculated with leprous material from 87 cases

of the disease. The media include those of most service in the culture of the tubercle bacillus and of fungi. Among the materials employed were oysters, the flesh of fish, cerebral matter and even human tissues provided by accidents, vitamins, and extracts of divers organs of man and of bovine animals. Both aerobic and anaerobic methods were used and the *p*H of the media varied from 6·5 to 8·5. Apparent multiplication may result from disintegration of inseminated globi, but subcultures could not be obtained from them. Macroscopical cultures of acid-fast bacilli were obtained in three instances, but one was identified as a human tubercle bacillus of feeble virulence, the other two were chromogenic paratuberculosis bacilli avirulent to guineapigs, etc. In addition, after eleven months, a bacillus believed to be that of Hansen was obtained, but subcultures did not produce visible colonies. The composition of the medium used in the last case will be published in a future paper. *L. R.*

LOVING (Walker L.). **Experimental Infection of the Rabbit with Duval's Chromogenic** *B. leprae* **Culture.**—*Proc. Soc. Experim. Biol. & Med.* 1941. Feb. Vol. 46. No. 2. pp. 293–295.

——. **The Experimental Infection of Rabbits with Duval's Chromogenic Acid-Fast Bacillus from Human Leprosy.**—*Jl. Infect. Dis.* 1941. May–June. Vol. 68. No. 3. pp. 193–206. With 8 figs. [10 refs.]

These two papers deal with the same work, the second one in greater detail and with histological illustrations. The author reports the successful infection of rabbits by 2 cc. doses of a heavy suspension of cultures of Duval's chromogenic bacillus of human leprosy, but negative ones with two other acid-fast bacilli, *Myco. phlei* and *Myco. smegmatis.* The injections were made subcutaneously, intraperitoneally or intravenously, and repeated twice at weekly intervals. The animals were killed and examined at varying intervals and it was found that cultures were increasingly difficult to obtain in direct proportion to the length of the sojourn of the organism in the animal's tissue. While the gross lesions in the rabbits were not typical of human lesions, the tissue microscopical changes were identical, with lymphoid, epithelioid and lepra or foamy cells. The infection was progressive over six to eight months, during which the micro-organisms steadily increased in numbers. *L. R.*

ITAKURA (Teiju). **The Histo-Pathological Studies on Teeth of the Lepers, especially on its Gingiva and other Supporting Tissues.**—*Japanese Jl. Med. Sci.* Pt. V. Pathology. 1940. Oct. Vol. 5. No. 3. pp. 201–220. With 11 figs.

[See this *Bulletin*, 1941, Vol. 38, p. 218.]

LOWE (John) & CHATTERJI (S. N.). **The Onset of Leprosy of the Lepromatous Type.**—*Leprosy in India.* 1941. Jan. Vol. 13. No. 1. pp. 14–17.

The authors discuss the relative frequency with which lepromatous cases on the one hand commence with nerve symptoms, and on the other are lepromatous in nature from the first. The histories and clinical examination of 249 lepromatous cases

indicate that between 65 and 68 per cent. started with lesions suggestive of the neural type of leprosy, and subsequently developed into definitely lepromatous cases. In over half of these the original lesions were caused by nerve trunk involvement, nearly one-third were in the form of patches with little or no erythema and thickening, and the remainder, about one-seventh, showed the definite erythema and thickening usually associated with the more marked tuberculoid lesions. In view of the frequency of tuberculoid cases in Calcutta these findings probably indicate that major tuberculoid lesions rarely become lepromatous. The average duration of the neural phase is 3–4 years, and long-standing neural cases rarely become lepromatous. *L. R.*

LOWE (J.) & CHATTERJI (S. N.). **The Development of Leprosy in a Child.**—*Leprosy in India.* 1941. Apr. Vol. 13. No. 2. p. 68. With 1 plate.

The patient was one of a family of eight persons—a mother and seven children. The father had died of leprosy and though the mother was healthy, five of the children showed signs of the disease. In 1933, at the age of five, the patient showed little more than depigmented patches on the buttocks, knees, face, hands and forearms, but in 1940 there was diffuse lepromatous infiltration of the skin of the whole body, some nodulation of the ears and many thickened nerves. This is an example of a common course of events. *C. W.*

LOWE (J.) & CHATTERJI (S. N.). **An Abortive Case of Leprosy.**—*Leprosy in India.* 1941. Jan. Vol. 13. No. 1. p. 30.

During recent years the frequency with which leprosy may be abortive has been increasingly emphasized and such cases are common in India. Most of the lesions are of the neural type. The authors describe the case of a man who in 1933 showed only slight thickening and tenderness of the ulnar nerve, without loss of sensation or anaesthetic patches. X-ray showed that this was not due to a cervical rib and on exposing the nerve and opening its sheath smears were made which were found to contain a few acid-fast bacilli. After the operation the slight deformity, which had been found in the bending of the little and ring fingers, disappeared and there have now been no signs of the disease for seven years. It is not thought that this result can be attributed to the operation, but that it is rather an instance of spontaneous arrest. *C. W.*

LOWE (J.) & CHATTERJI (S. N.). **An Acute Onset of Leprosy of Lepromatous Type in a Man aged Seventy.**—*Leprosy in India.* 1941. Jan. Vol. 13. No. 1. p. 29. With 1 plate.

In India leprosy rarely appears late in life, and when it does, is usually of a mild form. The authors describe the case of a man of 70 who noticed numbness in his right foot and leg, followed four months later by the appearance of thick red patches on the thigh, face, ears, body and extremities. Both peroneal nerves were thickened and there were patches of anaesthesia. Smears from the face, ear, arm and

thigh showed acid-fast bacilli, and a lesion excised for biopsy proved to be a fairly typical leproma. The clinical findings support the patient's story, the lesions all being obviously fairly new and acute.

C. W.

VELASCO (Felix). **Tuberculoid Leprosy; its Transformation to the Lepromatous Type.**—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 91–100. With 13 figs. on 3 plates. [18 refs.]

The author records and illustrates the naked eye and microscopical changes in two cases of tuberculoid leprosy which developed into the lepromatous type, contrary to the contention of some recent writers. In the first the interval was from 1933 to 1939, and in the second from 1935 to 1940. Such cases are rare, and he suggests that life-long studies of cases beginning in childhood are necessary for the study of the evolution of the disease.

L. R.

SARDJITO. Bacterioscopische lepra diagnose met daarmede samenhangende beschouwingen. [**Bacterioscopic Diagnosis of Leprosy.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1941. Apr. 1. Vol. 81. No. 13. pp. 739–746.

DHARMENDRA & BOSE (R.). **Complement-Fixation in Leprosy with Antigens prepared from Various Acid-Fast Bacilli.**—*Indian Jl. Med. Res.* 1941. Jan. Vol. 29. No. 1. pp. 7–21.

"1. The work was undertaken to find out whether complement-fixation tests done with sera from cases of leprosy and with antigen prepared by modern methods from the so-called cultures of leprosy, would give any evidence regarding the relation of these isolated organisms to the disease.

"2. Complement-fixation tests have been performed in 112 cases of leprosy and 58 cases of other diseases in presence of antigens prepared from six different acid-fast bacilli including the so-called leprosy bacilli of Duval, Bayon, Kedrowsky and Lleras. All the antigens were prepared by the method by Witebsky, Klingenstein and Kuhn.

"3. Sera diluted 1 in 5 fixed complement in presence of all the six antigens, in 84 per cent. of the 19 cases of the 'lepromatous' type, 61·5 per cent. of the 13 bacteriologically positive cases of the 'neural' type and 22·5 per cent. of the 80 bacteriologically negative cases of the 'neural' type and all the eight cases of leishmania infection.

"4. In 1 in 5 dilution 21 per cent. of the 14 Wassermann positive sera and 30 per cent. of the 20 sera from cases of leucoderma fixed complement in presence of one or more but not all the antigens.

"5. With sera diluted 1 in 25, complement-fixation is practically limited to cases of leprosy and kala-azar. In this dilution, however, the number of reacting sera of the 'neural' type of leprosy is reduced markedly.

"6. All the six antigens appear to behave in a similar way but the antigen prepared from the Lleras' bacillus appears to be slightly more sensitive. A slightly higher number of sera fix complement in its presence and dilution does not reduce complement-fixation with this antigen to the same extent as with the other antigens.

"7. This greater sensitivity of the antigen prepared from Lleras' bacillus does not appear to be caused by any specificity as it is seen both in leprosy and non-leprosy cases.

"8. It is concluded that complement-fixation tests have not given, and are unlikely to give, any evidence regarding the genuineness of cultures of organisms isolated from leprous lesions."

EAGLE (Harry), HOGAN (Ralph B.), MOHR (Charles F.) & BLACK (Samuel H.). **On the Reactivity of the Serum and Spinal Fluid of Leprous Patients with Spirochetal Suspensions.**—*Amer. Jl. Syph.* 1941. July. Vol. 25. No. 4. pp. 397–405.

The authors confirm and extend the observation of CAPPELLI that leprous cases give negative complement-fixation tests for syphilis with a suspension of cultured spirochaetes (*Spirochaeta pallida*, Reiter strain) as an antigen instead of a mammalian tissue extract. The frequent positive Wassermann tests in non-syphilitic leprosy patients can thus be differentiated from true syphilitic reactions in which there is response to both antigens. Thus, of 37 lepers giving positive flocculation tests, 25 of whom were also positive to the Wassermann test, all but six were negative to complement fixation tests. Moreover, the serological reactivity of Wassermann-positive syphilitic and leprous sera differed in that the latter tended to give a disproportionately high titre in a Wassermann as compared with a flocculation test. This was found to be the case in three of six positive spirochaetal reactions in leprosy patients, so only the remaining three were probably syphilitic. The spinal fluids obtained from nine leprous patients were completely negative to all the tests used. *L. R.*

COCHRANE (R. G.), RAJAGOPALAN (G.), SANTRA (I.) & RAJ (M. Paul). **A Study of the Lepromin Reaction in Children with Special Reference to Contact.**—*Leprosy in India.* 1941. Jan. Vol. 13. No. 1. pp. 5–13.

The authors report on lepromin tests in 276 Madras children and in 471 inmates of the Lady Willingdon Leprosy Sanatorium. Readings were taken once a week for six or more weeks, and reactions measuring less than 5 mm. were considered to be of little significance. Tables record the reactions in different types of the disease. Special attention was paid to the question of contact with leprous persons in relation to the development of reactions. In discussing this the invariably negative lepromatous cases should be excluded, and the conclusion is arrived at that the lepromin reaction tends more often to be negative in those in whom a history of contact is maximal, but the reaction is not significantly influenced by hereditary predisposition. The most important factor in breaking down cellular resistance in leprosy is continuous contact with an open case. *L. R.*

WADE (H. W.). **The Lepromin Reaction in Normal Dogs ; Preliminary Report.**—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 39–56. With 1 folding fig. [20 refs.]

The author confirms and extends the observations of RODRIGUEZ that the dog reacts positively to intracutaneous injection of lepromin, and therefore offers a convenient medium for a study of the Mitsuda reaction.

The curve of the reaction shows an immediate nonspecific oedematous reaction with recession in two days. After a variable period of latency, usually of about seven days' duration, occurs the onset of the definitive reaction consisting of three phases: primary development, height of activity on the average at the end of the third week, and finally recession in six to eight weeks. Reinjections have a disturbing effect in the second unstable period, but not in the recessive stage. It is concluded that this reaction, while of an allergic nature, is not one of hypersensitiveness, but rather one of capability of developing an allergic state after the introduction of the antigen. The specific feature of leprosy is the loss of that capability in lepromatous cases. *L. R.*

COLLIER (D. R.). **The Use of Diphtheria Toxoid in the Treatment of Leprosy. Second Report.**—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 1–10. With 6 figs. on 1 plate.

This is a further report from Thailand on the trial of injections of diphtheria toxoid in leprosy [see this *Bulletin*, 1941, Vol. 38, p. 26]. Over 600 leprosy cases have now been treated for periods ranging between a few weeks to 10 months, and it is claimed that the "results far exceed any obtained by me with any other method or combination of methods." In early cases treated for six months or more 50 per cent. have become symptom-free as judged by bacteriological examination, the condition of the skin lesions, areas of anaesthesia, and the patient's general condition. The more advanced cases show definite improvement in a high percentage of cases according to the same standards. Photographs of two cases showing great improvement are given [see below for other less favourable trials]. *L. R.*

MOISER (B.). **Report on Trial Treatment of Leprosy with Diphtheria Anatoxine Ramon.**—*Leprosy Review.* 1941. July. Vol. 12. No. 3. pp. 54–56.

This experienced worker reports on 19 cases of leprosy treated with diphtheria anatoxin in Southern Rhodesia. He began with 1 cc. weekly, and increased by 1 cc. to a maximum of 4 cc. weekly, each increase being made on the average at the end of six weeks. All the patients complained of "pains all over the body" and particularly in the knees and ankles. The experiment was finally stopped at the request of the patients and because the majority did not show any improvement. An analysis of the 19 cases shows that six were worse, and 11 showed no improvement or no change. Of the remaining two one showed ulceration and disappearance of a few of the nodules, but the general mass of the lesions remained unaffected and there was no improvement in the neural signs, so that any improvement was slight. The remaining case showed only a few small lepromata, which disappeared, so definite improvement took place, but the author observes that she was the type of case that improves rapidly under moogrol. [The reviewer has received unpublished reports from Dr. E. MUIR and others in which the results were uniformly unsatisfactory and completely failed to confirm the claims of the Thailand (Siam) workers, whose last report only claims 50 per cent. of recoveries in early cases, or much fewer than Moiser and others have recorded from the use of chaulmoogra preparations in that class of case.] *L. R.*

DAVISON (A. R.). **Blood Transfusions in the Treatment of Leprosy.**—*Leprosy Review.* 1941. Apr. Vol. 12. No. 2. pp. 32–40. With 12 graphs.

This careful trial of blood transfusions in leprosy at the Pretoria Institution in South Africa was carried out at the request of the seven European patients there. Six intravenous injections of from 350 to 400 cc. of blood were given at intervals of one month, after the necessary tests had been carried out by the Rand Blood Transfusion Service. Only one patient, whose first test proved to be erroneous, showed temporary untoward symptoms. It was hoped either to build up the patients' resistance or perhaps to stimulate their reticulo-endothelial system. Most of the cases were fairly advanced lepromatous ones with a bad prognosis. Periodical estimations were made of the sedimentation index, blood corpuscles, haemoglobin, clinical and bacteriological conditions, some of which are illustrated by charts. The results were disappointing, as in only one case was fleeting benefit noted and in two the condition continued to grow worse. It has not yet proved possible to make use of the blood of arrested cases of leprosy.

L. R.

KUZNEZOV (V. N.). **Experience with the Naphthalan Oil Bath Treatment of Leprosy.**—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 23–28.

This is a report on the use of medicinal baths of naphthalan oil in the Caucasian regions of Russia, where alone it occurs. It adheres firmly to the skin and cannot be washed off with water. The leprosy patients are immersed in it up to the breast at a temperature of 40° [scale not mentioned] for 10 to 15 minutes, after which the oil is scraped off and the patient lies in the sun to produce profuse sweating. Two series of ten baths, with five to ten days' intervals between the two sets is advised, during which the patient does not wash. It is claimed that this treatment "gave stable effects in almost all cases," with more rapid improvement than with any other treatment [unspecified] used in the leprosarium. *L. R.*

BALIÑA (Pedro) & BASOMBRIO (Guillermo). **Removal of Solitary Lesions in Tuberculoid Leprosy.**—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 11–14. With 8 figs. on 2 plates.

In this report is tabulated information on 14 cases in which primary lesions of a tuberculoid nature, including one in which a nerve alone was involved, were removed. Relapse occurred in only one of 12 followed up for periods of from one year and seven months to eight years and eight months. In seven other cases tuberculoid lesions were destroyed by the galvano-cautery (in three), by electro-coagulation (in three) and in one a thickened nerve was also removed. None of these had relapsed in periods from two and a half to nine months. All the patients received in addition regular chaulmoogra treatment and the only relapse was in a patient who did not go on with the injections. Illustrations of some of the cases are given; the authors are favourably impressed with the results so far obtained. *L. R.*

MUIR (E.). **A Note on the Treatment of Lepromatous Ulcers.**—*Leprosy Review.* 1941. Apr. Vol. 12. No. 2. pp. 40–41.

The author records that he found a surprisingly large number of lepromatous ulcers in the Chacachacare leprosarium of Trinidad, as compared with India, where trophic ulcers are by far the more common. They were due to breaking down of nodules and diffuse lepromata, with discharge of masses of lepra bacilli. A large part of the nursing sister's time was taken up by the dressings. He was led by reports of success in treating burns with silver nitrate, tannic acid and gentian violet to try these applications, and obtained highly satisfactory results in lessening the loss of time of the staff and the danger of spreading infection. He advises first painting with a 1 per cent. solution of gentian violet or methyl violet in alcohol, then with a 10 per cent. solution of silver nitrate in water, followed by a 15 per cent. watery solution of tannic acid three or four times on the first day; later usually one painting with tannic acid, with or without the use of a dye solution, suffices. Ulcers rapidly begin to heal up, including those on the lips or face, greatly to the comfort and cheerfulness of the patients. *L. R.*

SLOAN (Norman R.). **The Use of Urea in the Treatment of Perforating Ulcer.**—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 15–22. [11 refs.]

The author was led by a report on the value, in the treatment of purulent wounds, of a 2 per cent. solution, or a 15 per cent. ointment or jelly, to try a treatment of leprous perforating ulcers which consisted of daily packing with urea and instilling a saturated solution into sinuses. In 50 negro patients 80 per cent. healed, against 60 per cent. with other methods. *L. R.*

SEN (P.). **Leprosy. A Review of the Reports of Various National and International Conferences on Leprosy.**—*Calcutta Med. Jl.* 1941. Mar. Vol. 38. No. 3. pp. 139–143.

"Opinion is unanimous that leprosy is infectious, spread chiefly if not entirely by direct contact, and that leprosy can be controlled only by isolation of infectious cases particularly from children and young people, isolation measures however being augmented by treatment, propaganda and welfare work of various kinds."

DAVEY (T. F.). **A Repeated Leprosy Survey in Southeastern Nigeria. The Progress of Untreated Cases of Leprosy.**—*Internat. Jl. Leprosy.* Manila. 1941. Jan.–Mar. Vol. 9. No. 1. pp. 77–86.

This is an account of the progress of an untreated series of cases, between two surveys, in south-eastern Nigeria. With the cordial help of the chiefs a house-to-house survey was made in 1937 and repeated in 1939 in a large and very insanitary village, including a neighbouring leper village in which the people themselves had isolated the most advanced cases 10 years before. The incidence of the disease at the first survey was 6 per cent., raised at the second by the discovery of some additional cases, in persons absent at the first, to 6·7 per cent. Two years later the incidence had fallen to 5·7 per cent. apparently as the result of the village segregation, but 10 new cases had developed.

On comparing the full notes of the cases and of bacteriological examinations at each survey, and classifying them according to the Cairo Congress nomenclature, the progress during two years in the absence of treatment is shown in a table of 118 persons. Deaths amounted to 18, six of them from smallpox. Thirty-three patients were worse, 39 stationary and 28 had improved. Thus no less than 67 per cent. were stationary or had improved ; all but three of the improved cases were of the tuberculoid and simple macular neural type. The three improved lepromatous cases were macular ones. All the infectious patients have now been accommodated in the model leper village built by themselves under supervision, where they are living happily. It is hoped to make a further survey in two years time to ascertain the results of these measures, which now include regular treatment at a newly established clinic. *L. R.*

ROGERS (Leonard). **The Problem of Children born in Leprosy Colonies and Villages.**—*Leprosy Review.* 1941. July. Vol. 12. No. 3. pp. 50–53.

The author points out that among backward races of Africa and Korea male leprosy patients will refuse to enter colonies unless accompanied by their wives. This results in children being born to them, who are gravely exposed to infection from their parents, thus maintaining the incidence of the disease. He draws attention to the successful solution of this problem in the Korea settlement under Dr. R. M. WILSON through voluntary sterilization of the male partner and suggests its more general adoption under such circumstances. [See this *Bulletin*, 1935, Vol. 32, p. 858, and 1937, Vol. 34, p. 602.] *L. R.*

MALARIA.

PRÉCIS OF ABSTRACTS IN THIS SECTION.

CASTELLI (p. 708) reports on relapses which have occurred in men repatriated to Italy from East Africa. Although most of the infections in that country are due to *Plasmodium falciparum*, the relapses observed were almost entirely due to *P. vivax*. BRAMBILLA (p. 709) states that in Dirre Daua malaria is only slightly endemic, and that it is due more to *P. vivax* than *P. falciparum*. He notes that relapsing fever is relatively common, and incidentally that *Aëdes aegypti* is widely diffused. Anophelines are few in numbers, but *A. gambiae* is found. MOISE (p. 709) reports on malaria in Assab, Eritrea.

PAMPANA and CASINI (p. 710) report on an epidemiological study in Sardinia, where *A. maculipennis labranchiae* is the vector. The first wave of subtertian fever in the epidemic season, in July, is due to new infections, the number of crescent carriers before this time being sufficient to account for the outbreak.

FARID (p. 710) shows that on the eastern side of the Nile delta *A. sergenti* is commonly found and is frequently infected ; *A. pharoensis*, usually regarded as the vector, is rare and is not commonly infected. Subtertian malaria is the predominant variety. SEN (p. 711) reviews the anophelines of Assam. *A. minimus* is responsible for the intense malaria of the foothills, *A. philippinensis* for that of the

upper deltaic regions, and *A. sundaicus* for that of the coastal area. *A. culicifacies*, though widespread, appears to be of no importance as a carrier.

LIEM TJAIJ TIE (p. 711) reports seven cases of congenital malaria in the Dutch East Indies, and PIK GING HOO (p. 711) one from Amsterdam. Details of the latter are given.

NARAYAN (p. 712) records a case of cerebral malaria in which meningitis was simulated.

DE LEON (p. 712) reports favourably on a preparation of sulphanilamide in subtertian malaria.

GRANETT (p. 713) describes a method of estimating the repellent power of chemicals for mosquitoes, and describes the effects of a proprietary preparation which was found to be considerably more useful than citronella oil and certain other preparations.

RITA (p. 714) notes that chick embryo cannot be infected with *P. gallinaceum.*

TALIAFERRO and TALIAFERRO (p. 714) show that, in chickens, immunity acquired against *P. lophurae* can be passively transferred provided sufficient doses of immune serum are used and continued over a sufficient period.

DAS GUPTA and SIDDONS (p. 715) describe a Plasmodium and a trypanosome from the Munia. *C. W.*

BRAMBILLA (A.), MARA (L.) & CAMPISI (P. Marino). Istituzione di un centro di studi in A.O.I. [**A Research Centre in East Africa.**]—*Riv. di Malariologia.* Sez. I. 1940. Vol. 19. No. 5. pp. 336–344. With 6 figs. English summary.

"The 'Ettore Marchiafava' Malariology Institute has extended its activity to the Italian East Africa territory. After a preliminary survey carried out in 1936–1937 many fundamental epidemiological data have been collected setting in evidence the need of carrying out systematic studies in several zones to be exploited. During the following years, the procedure was fixed with the Italian Africa Ministry and with the General Government of Italian East Africa in view of establishing a Research Centre of the Institute in Africa. In 1939 three Research Stations have been organized, namely at Dire Daua (Harar), Agordat (Erythrea) and Genale (Somaliland).

"Descriptions are given of the Stations, of the equipment and of the area under control."

CASTELLI (G. D.). Le recidive malariche nei reduci dall' Africa Orientale. [**Malaria Relapses among Men Repatriated from East Africa.**]—*Riv. di Malariologia.* Sez. I. 1940. Vol. 19. No. 5. pp. 310–317. [27 refs.] French summary (9 lines).

Between January 1937 and August 1939 eighty-eight patients were admitted to the Military Hospital of Padua suffering from relapses of malaria, the infection of which had been acquired during service in East Africa. These cases were studied with special reference to the intervals that had elapsed since the date of infection. The author concludes that febrile relapses are rare after 15 months from the date of infection, and exceptional after two years. *P. vivax* was responsible for a ten-fold greater number of these relapses than was *P. falciparum*,

though infection with the latter is most common in the East African territories in which infection was acquired. No patients with relapses of *P. malariae* infection were admitted to hospital during this period.

Norman White.

BRAMBILLA (A.). Il problema della malaria a Dire Daua. [**The Problem of Malaria in Dirre Daua.**]—*Riv. di Malariologia.* Sez. I. 1940. Vol. 19. No. 5. pp. 290–309. With 7 figs. English summary.

Dirre Daua is situated at a height of about 1,200 metres. The European and native quarters of the town are separated by a river bed. The population of the former was about 3,500 plus a variable number of Italian troops. The population of the native quarter was about 15,000, a mixed and in part a nomadic population. When the author assumed charge of the local Malaria Research Station in 1939 he was asked to make an immediate study of the malaria situation; malaria was said to have been extremely prevalent the previous year. The results of his investigations are described.

Malaria is only mildly endemic in Dirre Daua. In the past other diseases have been responsible for errors in diagnosis. At certain seasons of the year dengue is very prevalent; *Aëdes aegypti* is widely diffused. The blood of 973 patients, in whom the symptoms justified a suspicion of malaria infection, was examined; in only 239 of these were malaria parasites found. In 9 per cent. of the total, spirochaetes of relapsing fever were found, mostly in October and November. Further evidence of the moderate endemicity of malaria is given by the spleen index, 17 per cent., and the parasite index, 15·6 per cent., of 835 children under 14 years of age. Of the positive blood smears from these children 66 per cent. contained *P. vivax*, and 34 per cent. *P. falciparum*.

Adult anophelines were found only in native huts and even there only in limited numbers. A few *A. gambiae* could be found at all times of the year. Other species found were *A. d'thali*, *A. pretoriensis*, *A. cinereus* and *A. turkhudi*. The only important breeding places were found in the bed of the Harré torrent which is three kilometres east of the native town. Prophylactic measures, including antilarval measures and mechanical protection of dwellings, based on the results of this inquiry, were put into operation and had already achieved encouraging results.

N. W.

MOISE (Regolo). Osservazioni sulla malaria di Assab e sull'anofelino che la trasmette. [**Observations on Malaria in Assab and on the Anopheline which transmits it.**]—*Riv. di Malariologia.* Sez. I. 1940. Vol. 19. No. 3. pp. 149–158. With 5 figs. English summary.

The observations recorded refer to the period December 1935 to April 1936. During this period among the Italian naval personnel, averaging about 500, in Assab, Eritrea, there were 25 cases of malaria, 15 *P. vivax* and 10 *P. falciparum* infections. The adult native population appears to suffer but little from malaria. The spleen index of 97 native children was 41 per cent. and the parasite index of 85 children

35 per cent. Of the 30 positive blood smears from children, 21 contained *P. falciparum*, 8 *P. malariae* and 1 *P. vivax*. The only anopheline found was stated to be *A. culicifacies*: it was found breeding in wells and cisterns. [*A. culicifacies* has not been found by other workers in East Africa. *A. culicifacies* var. *adenensis* has been reported by CHRISTOPHERS from Southern Arabia only; there is no record of transmission by this variety, see EVANS, *Mosquitoes of the Ethiopian Region*, Part 2, 1938, pp. 172–174]. *N. W.*

PAMPANA (Emilio) & CASINI (Guido). Studi di epidemiologia malarica in Sardegna. [**Studies of Epidemiology of Malaria in Sardinia.**] —*Riv. di Malariologia*. Sez. I. 1940. Vol. 19. No. 5. pp. 273–289. With 6 figs. [12 refs.] German summary.

In order to study the genesis of the annual epidemic manifestations of malignant tertian malaria in Sardinia two small adjacent villages, Loiri and Montelittu, were selected, with populations of 137 and 97 respectively. During the period of observation neither antilarval measures nor the prophylactic administration of drugs nor any other measure to protect the populations from infection were undertaken. In febrile cases of malaria five-day courses of quinine were given. The blood of 36 inhabitants of Loiri and of 39 of Montelittu was examined twice a week from the 15th May to the 31st July. The observations began in Loiri on the 1st September, 1938, and in Montelittu on the 25th March, 1939, and were continued till the 15th December, 1939. All epidemiological data were carefully recorded.

The anophelines found were *A. claviger*, *A. algeriensis* and *A. maculipennis labranchiae*, the last-named being the vector.

The observations showed that the first wave of cases of malignant tertian fever, at the beginning of the epidemic season in July, were new infections and not relapses. In the pre-epidemic season the number of crescent carriers, though small, was sufficient to account for the subsequent outbreak. It was noted that hibernating female *A. maculipennis labranchiae* begin to leave their winter quarters at the end of February and the first generation of adults hatches out in April. There appeared to be a relationship between the density of *A. m. labranchiae* and the mean weekly temperature: mean temperatures below 15°C. and above 25°C. appear to be unfavourable for this insect.

N. W.

FARID (Mohyddin). **Malaria Infection in** *Anopheles sergenti* **in Egypt.**— *Riv. di Malariologia*. Sez. I. 1940. Vol. 19. No. 3. pp. 159–161.

The author studied an outbreak of malaria in two villages in the Sharqia Province, on the eastern side of the Nile delta. Parasite rates were up to 70 per cent. (with great predominance of *Plasmodium falciparum*). Larvae of *Anopheles sergenti* were found in numbers in spots where water seeped into a large drain, and those of *A. pharoensis* (generally regarded as the vector in the Nile valley) were rare. Adult *A. sergenti* were common in rooms and in rough stables, and six out of 220 had gland infections (sporozoite rate 2·7 per cent.). This mosquito has not previously been recognized in the Nile valley, though known to occur in the oases in the Western Desert, in the Canal Zone, and Sinai. The author has made over 1,700 dissections of *pharoensis* from various parts of the delta, and found no positives.

[These interesting observations are consistent with what is known or suspected elsewhere, that *A. sergenti* is easily overlooked, but important as a malaria carrier.] *P. A. Buxton.*

SEN (P.). **Malarial Mosquitoes of Bengal.**—*Bengal Public Health Jl.* 1940. Dec. Vol. 2. Nos. 2 & 3. pp. 87–94. With 2 plates. [14 refs.]

This is a concise review of the relation of Anopheles to malaria in Bengal. The province consists of six zones: montane, submontane, lateritic moor country, upper deltaic, lower deltaic and estuarine. There are thirty-one species of Anopheles in Bengal but only three, *A. minimus*, *A. philippinensis* and *A. sundaicus* are important as malaria carriers. The montane region suffers little from malaria. The hill streams of the submontane area, the terraced rice-plots, and the drains of the tea gardens provide the breeding places of *A. minimus*. This area is intensely malarious. In the drier lateritic zone there is little malaria. But in the upper deltaic region "tanks", ponds, silted-up beds of rivers and the low-lying marshes provide the breeding places of *A. philippinensis* and in some districts endemic malaria is severe. In the lower deltaic region *A. philippinensis* is again responsible for some of the malaria, but most is due to *A. sundaicus* breeding in "fish ponds" or rice fields from which the sea water has been excluded by embankments. In the estuarine area the clearance of mangrove in recent years has led to a great increase in the breeding of *A. sundaicus*, which is the species responsible for the increased malaria in the Sunderbuns and the Salt Lake area near Calcutta. *A. culicifacies*, though widespread in Bengal, seems to be of no importance there as a malaria carrier. [See also WHITE, this *Bulletin*, 1941, Vol. 38, p. 505.]

V. B. Wigglesworth.

LIEM TJAIJ TIE. Over aangeboren malaria. [**Congenital Malaria.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1941. Jan. 28. Vol. 81. No. 4. pp. 185–192. [12 refs.] English summary.

"A description is given of seven cases of congenital malaria in three native and three Chinese new-born infants and one Indo-European child (4 girls and 3 boys).

"Six of the children had sub-tertian and one tertian parasites in their blood.

"The parasites were found as follows: directly after birth, on the 2nd, 11th, 17th, 24th, 26th and 34th day of life. Of the latter five children three were born in the hospital and had not been home when the parasites were found. Two of the other children were brought to the hospital shortly after birth. In three of the children the margin of the spleen could be felt (from 1 to 1½ fingers breadth below the ribs).

"With a probability amounting to certainty the malarial infection was in all seven children intra-uterine.

"Congenital malaria is regarded in the literature as a rare condition, and also in the Netherlands Indies, in spite of the fact that malaria is still found almost everywhere, it is but seldom observed."

PIK GING HOO. **Congenital Malaria.**—Reprinted from *Acta Paediatrica.* 1941. Vol. 28. pp. 293–302. With 1 fig.

This case of congenitally-transmitted malaria is of considerable interest mainly because the proof is fairly conclusive and because the

case was one of indigenous benign tertian malaria originally contracted in the northern district of Amsterdam. Comparatively few instances of this kind have previously been reported in Holland. The exact mechanism by which transplacental infection of blood protozoa may take place is still a matter of conjecture. Some have considered that there must be some mechanical tear or damage so that a sharp separation between maternal and foetal blood no longer exists. It is possible, as is suggested here, that the malarial infection induces pathological changes in the placenta itself, thus facilitating passage of the parasites, or it may be that it takes place through a placental haemorrhage.

It is obvious that before a genuine case of transplacental infection can be obtained it is necessary that the child should be born in a locality where malaria is non-endemic and where no anopheline vectors abound. On the other hand, if such a case were to be observed in an endemic area it should occur at a time of the year during which potentially infective anophelines are absent. A further provision may be stipulated that the interval elapsing between birth and demonstration of parasites in the blood should be considerably shorter than the known incubation period.

In August 1940, an infant, 14 days old, was admitted to the Pediatric Clinic in Amsterdam with a suspicion of quinine poisoning as the mother suffered from severe postpartum recurrence of malaria (benign tertian) originally contracted in Amsterdam and for which she had taken large doses of quinine. The child, which was also found to be suffering from congenital heart disease and *icterus neonatorum*, had slight fever. At the age of 20 days ring forms of *P. vivax* were found in the blood and on the 24th day large numbers of gametocytes. Death took place some 80 days after birth presumably due to double *otitis media*. Diffuse malarial pigmentation was present in the liver and spleen.

There is one loophole in the evidence ; namely that the infant may have been bitten by an infected Anopheles mosquito after birth, but on several grounds this is ruled out as being unlikely. *P. Manson-Bahr.*

NARAYAN (Jagdish). **Cerebral Malaria simulating Meningitis.**—*Indian Med. Gaz.* 1941. Feb. Vol. 76. No. 2. p. 94.

In this case the cerebrospinal fluid was under pressure, but clear, the signs of meningitis were present and *P. falciparum* was found in the blood. Quinine sulphate, both intramuscularly and by the mouth, failed to control the fever, but on the tenth day after admission atebrin was given and the patient became afebrile in 48 hours, after which he completely recovered. *C. W.*

DE LEON (Amonario Diaz). El paludismo y su tratamiento intravenoso por las sulfanilamidas. [**The Treatment of Malaria with Intravenous Injections of Sulphanilamide Preparations.**]—*Medicina.* Mexico. 1940. Nov. 10. Vol. 20. No. 375. pp. 551–558. [28 refs.]

The author discusses the relative merits and limitations of quinine, atebrin and plasmoquine in the prophylaxis and treatment of *P. vivax* and *P. falciparum* infections. In acute attacks of the latter sulphanilamide is a useful substitute for atebrin in special cases in which

intravenous medication is indicated. Sulphanilamide is exclusively schizonticidal. Five illustrative cases are described. The sulphanilamide preparation used was ambesid. *N. W.*

CHOPRA (R. N.), HAYTER (R. T. M.), SEN (B.) & TALUKDAR (M.). Crinodora (Palusan) nella lotta contro la malaria in India. [**The Use of Crinodora in Malaria in India.**]—*Riv. di Malariologia.* Sez. I. 1940. Vol. 19. No. 4. pp. 251–255. English summary (7 lines).

See this *Bulletin*, 1941, Vol. 38, p. 34.

GRANETT (P.). **Studies of Mosquito Repellents, I. Test Procedure and Method of evaluating Test Data.**—*Jl. Econom. Entom.* 1940. Vol. 33. No. 3. pp. 563–565. With 2 figs. **II. Relative Performance of Certain Chemicals and Commercially Available Mixtures as Mosquito Repellents.**—*Ibid.* pp. 566–572. [Summarized in *Rev. Applied Entom.* Ser. B. 1941. Apr. Vol. 29. Pt. 4. pp. 65–66.]

"The first paper contains a description of the procedure used in investigations begun in New Jersey in 1935 for testing the repellency of chemicals to mosquitos in the field in conditions as near normal as possible and the method of evaluating the results. An untreated arm or leg is exposed to biting for a definite period, usually 2 minutes. A measured quantity of the chemicals to be tested is then applied to the other arm or leg, and it is exposed for the whole period of the test while the untreated limb is exposed for 2 minutes, at intervals of ¼ to ½ hour. The average number of bites per minute on the untreated area is called the "biting frequency" during the test period. The time in minutes required to obtain the first bite on the treated area is called the "repellent time." This time is the customary measure of the effectiveness of a repellent, but changing factors in the field cause it to fluctuate. An increase in biting frequency is correlated with a decrease in repellent time. Curves are given which show this relationship for four repellents. For any pair, the difference between their repellent times at a given biting frequency is approximately constant. At the highest biting frequencies, the decrease in repellent time is relatively slight. For purposes of comparison, the most satisfactory product developed during the investigation and known under the brand name of Sta-Way Insect Lotion is used as a standard. The "repellent rating" of a material is its repellent time expressed as a percentage of that of the standard at the same biting frequency. If the product is compared with the standard at several biting frequencies, the repellent ratings are averaged.

"In the second paper, the literature on repellents is reviewed and the properties desirable in an ideal one are given. As none was available possessing anything approaching the qualities named, the investigations previously referred to were begun to discover or develop a better substance. Nearly 1,000 selected organic chemicals and chemical mixtures and about 40 proprietary products were tested, chiefly against *Aëdes sollicitans*, Wlk., and *A. cantator*, Coq., and the highly satisfactory one referred to in the previous paper was developed. Its active ingredients are diethylene glycol monobutyl ether acetate and diethylene glycol monoethyl ether and represent 65 per cent. of the whole, and it also contains ethyl alcohol, maize oil and perfume. Its repellent time (67–136 minutes at biting frequencies of 26–1) was considerably longer than that of any other usable compound tested, it has a pleasing odour,

does not affect natural fibres, is non-irritant and not greasy or unsightly on the skin, and is effective against many other blood-sucking pests, though usually not for so long as against mosquitos. On the other hand, it injures paints, varnishes, lacquers and certain plastics and affects cellulose-acetate fibre. The four products that ranked next to it in effectiveness were a proprietary product containing citronella oil, one containing synthetic organic repellents, undiluted citronella oil and medium-fraction steam-distilled pine oil. Their repellent ratings were respectively 80, 66, 65 and 59 per cent. The superiority of the proprietary product over undiluted citronella oil is attributed to its excessively viscous properties. The best of the pyrethrum mixtures tested had a repellent rating of only 41 per cent., and moreover, during part of the time for which the mosquitos were repelled from sucking blood they used to alight on the limb, pierce the skin and fly away without feeding. This piercing of the skin is almost as annoying as an actual bite."

COVA-GARCIA (Pablo). La legislación antimalárica venezolana y proyecto de reglamentación. [**Anti-Malaria Legislation in Venezuela.**]—*Publicaciones de la Division de Malariologia.* Ministerio de Sanidad y Asistencia Social. Caracas. 1940. Dec. 13. No. 6. 34 pp. [Bibliography.]

RITA (Geo.). Tentativi di infezione dell'embrione di pollo con *Plasmodium gallinaceum.* [**Attempts to infect Fowl Embryos with** *P. gallinaceum.*]—*Riv. di Malariologia.* Sez. I. 1940. Vol. 19. No. 4. pp. 230–233. English summary.

"The author has tried experiments for artificially infecting chick embryos with *Plasmodium gallinaceum.* He has always obtained negative results, confirming what had formerly been found by V. CHORINE and by W. GAVRILOV and co-workers. On the contrary he was able to provoke a severe infection in newly hatched chicks. The reasons of this different behaviour are discussed."

TALIAFERRO (William H.) & TALIAFERRO (Lucy Graves). **Active and Passive Immunity in Chickens against** *Plasmodium lophurae.*—*Jl. Infect. Dis.* 1940. Mar.–Apr. Vol. 66. No. 2. pp. 153–165. With 3 figs. [21 refs.]

The experiments recorded were carried out with a strain of *Plasmodium lophurae* in young chicks. The infection is a benign one in that recovery by crisis occurs. Thus a six-weeks-old chick receiving intravenously a dose of infected blood resulting in the presence in its blood next day of 33 parasites per 10,000 red cells showed a steady increase of parasites till on the fifth day 580 per 10,000 red cells were present. The crisis was reached at this stage so that 48 hours later only one parasite per 10,000 red cells was present. A few were found during the next 48 hours but none later. That parasites are still present in such recovered chicks was proved by the infectivity of blood up to as long as four months. During this latent period there is a high degree of immunity to superinfection. Serum from such latently infected chicks, if injected along with parasites and continued daily during the period of usual rise in number of parasites, definitely suppressed the

infection. This passive immunity is greater if in the first place the immune serum and parasites to be injected are, before injection, incubated together for some time. It was noted that chickens older than six weeks exhibited some degree of natural resistance to infection, and evidence was obtained to suggest that there may be some natural antibodies in some normal sera. The clear result of the experiments is the demonstration that acquired immunity can be passively transferred, provided sufficient doses of immune serum are used and continued over a sufficient period. *C. M. Wenyon.*

Das Gupta (B. M.) & Siddons (L. B.). **On a Plasmodium sp. of the Malay Chestnut-Bellied Munia** [*Munia Atricapilla atricapilla* (**Vieill.**)].—*Indian Med. Gaz.* 1941. Mar. Vol. 76. No. 3. pp. 148–150.

—— & ——. **On a Trypanosome of the White-Throated Munia.**—*Uroloncha malabarica* (**Linn.**).—*Ibid.* pp. 151–152. With 1 plate.

In these two papers the authors describe and illustrate in a coloured plate certain blood parasites of birds in India.

In the first is given a description of a malarial parasite of *Munia atricapilla atricapilla* which had been imported to India from Malaya. The parasite resembles very closely the classical *Plasmodium praecox* but differs in certain details such as the number of merozoites. Furthermore it is not inoculable to the Indian house sparrow, while the canary is only slightly susceptible. The white-throated munia (*Uroloncha malabarica*) is even more susceptible than the original host. As the classical form is readily inoculable to the sparrow and canary the authors consider that the present form is a variety, which they propose to name *Plasmodium praecox* var. *muniae*. It is reported that a haemoproteus and a trypanosome were encountered in *U. malabarica*, a toxoplasm in the sparrow and a microfilaria in *M. atricapilla atricapilla*.

In the second paper the trypanosome of *U. malabarica* is described in detail. Though it resembles in many respects *Trypanosoma hannai* of the domestic pigeon of India it is regarded as a distinct species, for which the name *Trypanosoma knowlesi* is proposed. *C. M. W.*

PELLAGRA AND ALLIED CONDITIONS.

Précis of Abstracts in this Section.

Díaz-Rubio (p. 716) refers to pellagra and other deficiency diseases in Madrid during the Spanish Civil War. Carruthers (p. 717) points out that in India pellagra is probably more common than is suspected.

Kooser and Blankenhorn (p. 717) investigated the diet in a rural area in Kentucky where pellagra had been endemic but from which the

condition has now disappeared, and in an adjacent mining area in which it still persists. The better diet in the rural area is ascribed to education in the use of available foodstuffs by members of the Frontier Nursing Service.

SMITH *et al.* (p. 718) describe a condition (dyssebacia) in pellagra, in which the mouths of the sebaceous glands are plugged with inspissated sebum, giving the skin a rough appearance. This is usually seen on the face, but there is another condition, phrynoderma, of the shoulders and extremities, which is also associated. Dyssebacia is readily cured by autoclaved yeast and crude extract of liver.

From a study of cases in China, HOU (p. 719) concludes that ocular lesions associated with pellagra are due to ariboflavinosis, while cheilosis is due to ariboflavinosis plus a minor deficiency of nicotinic acid. SYDENSTRICKER (p. 719) discusses the eye lesions associated with riboflavin deficiency, of which the earliest and most constant is superficial vascularization of the cornea, which may progress to interstitial keratitis. SPIES *et al.* (p. 720) discuss the symptoms of ariboflavinosis observed in children in an area of Alabama where deficiency diseases are common.

URABE (p. 720) found evidence of liver damage in a high proportion of pellagrous patients ; the damage is apparently proportional to the intensity of the various symptoms.

Cases of human black tongue are described by WILKINSON and by MIDDLETON and MILROY (p. 721), but whereas the condition in the patient referred to by the first author responded to nicotinic acid (although the diet had previously been good), that in the patient described by the others cleared up after the removal of a diseased tooth.

NAGANNA *et al.* (p. 721) describe a modification of a method of estimating nicotinic acid in the urine, and quote their findings in normal and pellagrous persons.

TROWELL (p. 722), in a discussion of pellagra and certain allied conditions observed in African children, considers that all these should be included under the heading of pellagra. He discusses the meaning of the terms "mosaic skin" and "crazy-pavement skin." MCKENZIE (p. 722) found "crazy-pavement skin" in conditions not associated with any other sign of malnutrition. It was rarely associated with signs of lack of vitamin A. *C. W.*

DÍAZ-RUBIO (M.). Estudios sobre la enfermedad de Casal (pelagra).—Nota previa. [**Studies in Pellagra : Preliminary Note.**]—*Revista Clinica Española.* Madrid. 1941. Feb. 1. Vol. 2. No. 2. pp. 161–162.

The author remarks that from 1936 to 1939 the food supply in Madrid was grossly deficient in quality and quantity. He discusses the aetiology of pellagra and states that "para-pellagrous" states were commonly seen in Madrid, characterized particularly by lesions of the nervous system. Other deficiency conditions were famine oedema, hemeralopia, hyperkeratosis and anaemias, while minor degrees of scurvy were common. There were no cases of beriberi in spite of the fact that the diet was deficient in vitamin B_1. The author promises further discussion of these matters in later papers. [See also this *Bulletin,* 1941, Vol. 38, p. 401.] *C. W.*

CARRUTHERS (L. B.). **Pellagra in India.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1941. July 3. Vol. 35. No. 1. pp. 21–34. [19 refs.]

As has been pointed out before, the paucity of records of pellagra in India is probably no measure of the incidence of the disease in that country. In the article under review notes of ten representative cases admitted to the Miraj hospital, Bombay Presidency, are given, cases chosen from a large series coming under observation during the past two years, most of which belonged " to the so-called subclinical group." These notes will serve to draw the attention of other medical practitioners in India, who may be unfamiliar with pellagra, to the kind of clinical picture they should be on the look out for.

H. S. Stannus.

DEWAN (John G.). **The Etiology of Pellagra.**—*Amer. Jl. Psychiatry.* 1941. Mar. Vol. 97. No. 5. pp. 1188–1193. [25 refs.]

This is a good summary of early views and recent work on the subject. It contains nothing new. *C. W.*

KOOSER (John H.) & BLANKENHORN (M. A.). **Pellagra and the Public Health. A Dietary Survey of Kentucky Mountain Folk in Pellagrous and in Non-Pellagrous Communities.**—*Jl. Amer. Med. Assoc.* 1941. Mar. 8. Vol. 116. No. 10. pp. 912–915. With 1 fig. [Summary appears also in *Bulletin of Hygiene.*]

Pellagra is still endemic in certain regions. Its occurrence is bound up with many factors determining the scale of living. The opportunity arose to investigate the social conditions in a rural area in Kentucky where pellagra was endemic and has now disappeared, along with an adjacent coal-mining area where endemic pellagra is still present. Both areas have a large amount of unemployment. The dietary survey was made in the late winter and spring, when the diet is at its worst. The cash income was considerably less in the rural than in the urban area, but the consumption of pellagra-producing foods (fat pork, beans and corn meal) was about equal in both areas. The consumption of pellagra-preventing foods was, however, greater in the rural area (half of the families owning some land), where livestock production was much greater than in the urban area. An important difference was in milk consumption. In the coal-mining area the amount of fresh cow's milk consumed was negligible compared with that of the rural area, and also less eggs, lean pork, and chickens were eaten. The better diet in the rural area accounts for the absence of pellagra, but the improvement in the diet of this area, where economic conditions are very poor, had to be accounted for. It is due to the advent of the Frontier Nursing Service in this community. The Nursing Service has greatly improved the health in regard to infectious, parasitic, and particularly nutritional diseases. Pellagra disappeared, without any economic improvement, because the population had been educated to choose the right foods, available in their particular district. The mere prescribing of a good diet or nicotinic acid is not the whole solution of the pellagra problem. *H. N. H. Green.*

SMITH (Susan Gower), SMITH (David T.) & CALLAWAY (J. Lamar). **Dysfunction of the Sebaceous Glands associated with Pellagra.**—*Jl. Investigative Dermat.* 1941. Feb. Vol. 4. No. 1. pp. 23–40. With 2 figs. [34 refs.] [Discussion pp. 40–42.]

In addition to typical dermatitis many pellagrins present a different type of skin lesion, involving the sebaceous glands, which occurs mostly on the face and is independent of any effects of radiant energy. This is characterized by the development of innumerable plugs of inspissated sebum which project from the orifices of the sebaceous follicles, imparting a rough appearance to the surface, which on palpation resembles shark skin or sandpaper. In the early stages these small plugs reflect the light and have been likened to sulphur flakes. W. H. WILSON has demonstrated that by drawing a microscope slide over the surface of the nose the "sulphur flakes" powder away and leave no greasy smear as in the case of a non-pellagrin. These lesions appear first on the *alae nasi*, spread over the nose, the lips, and, when advanced, on to the forehead and chin, eventually involving the whole face. The absence of natural oily sebum suggests that the condition is a deficiency type of asteatosis rather than seborrhoea. The term "dyssebacia" is applied to this syndrome.

The second type of sebaceous gland change is associated with vitamin A deficiency and is known as phrynoderma, or toadskin; it is rarely seen on the face, and is usually distributed over the shoulders and extremities. Rats maintained on a riboflavin-deficient diet developed sebaceous gland lesions and were cured with either synthetic riboflavin or autoclaved yeast. A further step was in the purification of the basic diet and vitamin B_1 was supplied as thiamin chloride, and carbohydrate as sucrose. This diet is referred to as G-free diet 25 and is taken as a basic diet for future comparison, lacking all the factors of the vitamin G (B_2) complex. When rats were placed on this diet they developed the same type and degree of sebaceous gland change as occurred on the riboflavin-deficient diet of Bourquin and Sherman. If the tail was removed at this time and the rat fed on autoclaved yeast (0·5 gm. per day) an immediate growth impulse occurred.

Not one of the synthetic factors when given singly to rats on a G-complex-deficient diet is sufficient to protect them from developing the sebaceous gland lesion described. If riboflavin is the only deficiency, then synthetic riboflavin alone will protect, but if all other factors of the G-complex are missing from the diet, pure riboflavin has little or no effect. Nor has vitamin B_6 alone, in the absence of other G-complex factors, any protective effect against sebaceous gland lesions.

In a statistical study of 512 pellagrins dyssebacia was observed more frequently in adults than in children, in males than in females. The incidence is highest in negro males, particularly in alcoholics with dementia. The outstanding histological change is hyperplasia of the sebaceous glands, the follicles being dilated and plugged with dry sebaceous material. Dyssebacia is readily cured by autoclaved yeast and crude extracts of liver. Vitamin B_1 and parenteral liver extract are ineffective. On the other hand, synthetic riboflavin has little effect in the active pellagrin who has not been treated with nicotinic acid.

It is concluded that dyssebacia results from a deficiency of some factor or combination of factors present in the vitamin G-complex. Aetiologically the lesions of dyssebacia appear to be quite like the

lesions produced in the rat, although histologically they appear to be quite different, the rat lesion being characterized by atrophy while in man there appears to be hypertrophy and hyperplasia.

P. Manson-Bahr.

HOU (H. C.). **Riboflavin Deficiency among Chinese. 2. Cheilosis and Seborrheic Dermatitis.**—*Chinese Med. Jl.* 1941. Apr. Vol. 59. No. 4. pp. 314–325. With 7 figs. on 2 plates. [12 refs.]

In a previous communication the author pointed out that among the cases admitted to the eye clinic for ocular manifestations of ariboflavinosis only a few showed other signs of the syndrome as described by American writers, such as angular stomatitis, cheilosis and facial seborrhoeic dermatitis. Cheilosis has, however, been noted to be very common among the poor Chinese of Shanghai and widespread among the children in a refugee camp near by. It appears to be of common incidence also in typical cases of pellagra, but then again is often dissociated from other signs of the syndrome. Among 10 pellagrins admitted to hospital cheilosis and glossitis were present in all, facial seborrhoea in four and some ocular manifestation in five. In these patients, while the dermatitis cleared up with nicotinic acid, the cheilosis and seborrhoea showed but little improvement until yeast powder or riboflavin was given.

Of 186 refugee children examined 82 (44·1 per cent.) exhibited cheilosis ; of these 82, however, only nine showed any ocular manifestations ; seven children had ocular lesions but no cheilosis. Among the whole 186 children in none was the seborrhoeic affection of the face noted. These facts, the author thinks, suggest that the ocular lesions are due to an ariboflavinosis, while cheilosis is due to an ariboflavinosis plus a minor deficiency of nicotinic acid. He finds also that the cheilosis and facial seborrhoea respond more slowly to treatment with riboflavin than the eye lesions.

H. S. S.

SYDENSTRICKER (V. P.). **Clinical Manifestations of Ariboflavinosis.**—*Amer. Jl. Public Health.* 1941. Apr. Vol. 31. No. 4. pp. 344–350. [38 refs.]

Riboflavin deficiency is characterized by photophobia, dimness of vision at a distance, cheilosis, seborrhoeic lesions around the nose and ears, and specific glossitis. The eye affections are important and instructive. The earliest and most constant feature is superficial vascularization of the cornea, which may progress to interstitial keratitis. It may even be that that form of keratitis which accompanies acne rosacea is also due to riboflavin deficiency. Therefore recognition of the early ocular signs of riboflavin deficiency offers a ready and easy method of identifying B Group avitaminosis at a stage when no other gross signs are present. The facial and labial lesions of ariboflavinosis largely contribute to the typical facies of pellagra, and together with the glossitis, constitute the picture of "*pellagra sine pellagra.*"

Thiamin, nicotinic acid, and riboflavin are components of coenzymes essential to the intermediate metabolism of carbohydrate ; when any of the three vitamins is exhausted from failure of replacement, or when there is a marked failure of balanced intake, the clinical picture of avitaminosis becomes apparent. It has now become possible

to correlate many observations on nutritional disease which have so far appeared contradictory. Ariboflavinosis is therefore, like pellagra and beriberi, a manifestation of B Group avitaminosis, and the specific signs probably result from complex disturbance of the coenzyme functions of the B vitamins as a group, to which is added failure of activity of riboflavin in intracellular oxidation. *P. M.-B.*

SYDENSTRICKER (V. P.). **The Clinical Manifestations of Nicotinic Acid and Riboflavin Deficiency (Pellagra).**—*Ann. Intern. Med.* 1941. Mar. Vol. 14. No. 9. pp. 1499–1517. [40 refs.]

SPIES (Tom D.), BEAN (William B.), VILTER (Richard W.) & HUFF (Nelwyn E.). **Endemic Riboflavin Deficiency in Infants and Children.**—*Amer. Jl. Med. Sci.* 1940. Nov. Vol. 200. No. 5. pp. 697–701. With 2 figs. (1 coloured) on 1 plate. [12 refs.] [Summary appears also in *Bulletin of Hygiene.*]

Amongst malnourished children in an area in Alabama in which deficiency diseases are endemic the lesions of riboflavin deficiency are most commonly seen. Out of 472 children, aged from 5 months to 14 years, of parents with deficiency diseases, 241 showed one or more of the prominent lesions, *viz.* cheilosis, linear fissures of the lip, and eye symptoms, such as bulbar conjunctivitis and failing vision. All the children were underweight and apathetic. The disease waxed and waned but was most in evidence in the spring and summer. All responded rapidly to synthetic riboflavin by mouth ; often a striking increase in growth followed. Treatment of the mother with riboflavin or substances rich in riboflavin, resulted in the cure of the nursing infant. Bacteriological examination of the mouth and eye lesions showed *Staph. aureus haemolyticus* in about 80 per cent. and *Strep. haemolyticus* in about 20 per cent. of cases. *Corynebacterium xerosis* was isolated from a large percentage of the eye lesions and in pure culture from Bitot's spots in all 5 cases showing these spots. All the mothers had taken very inadequate diets during pregnancy and lactation. The children, whether nursing or not, started the family food at about two months of age, choosing the foods desired and usually selecting biscuits, corn bread, gravy and syrup. The majority of children received only 35 per cent. of the estimated requirements of riboflavin.

H. N. H. Green.

URABE (Kiyosi). Studien ueber Pellagra in Chōsen. IV. Mitteilung : Ueber den Magensaft und die Leberfunktion bei Pellagra. [**Gastric Juice and Liver Function in Pellagra.**]—*Japanese Jl. Dermat. & Urol.* 1940. Nov. 20. Vol. 48. No. 5. p. 107.

The author investigated 42 patients with pellagra ; the liver function tests comprised tests for urobilin, urobilinogen, the combining power of camphor and glycuronic acid, the Takata reaction, the Azorubin S reaction and the Widal test. Most of the patients showed reduction in gastric acidity, only four being normal in this respect. In the other tests, 14 gave normal results for urobilin, 14 for urobilinogen, 30 with the Takata reaction, 14 with the camphor test, 14 with the Azorubin S reaction and eight with the Widal test. The remainder showed various degrees of abnormality. A high proportion, therefore, showed

evidence of liver damage and it was found that the extent of the damage was proportional to the intensity of the various symptoms.

C. W.

i. WILKINSON (P. B.). **Human Black Tongue treated with Nicotinic Acid.**—*Caduceus.* Hong Kong. 1940. May. Vol. 19. No. 2. pp. 100–102.

ii. MIDDLETON (W. Malcolm) & MILROY (Gavin). **A Case of Black-Tongue.**—*Brit. Dental Jl.* 1941. Sept. 1. Vol. 71. No. 5. p. 176.

i. Human black tongue is an uncommon condition, the main features of which are hypertrophy of the filiform papillae of the tongue, with grey or black pigmentation. The onset is insidious, it may persist for years and does not cause symptoms. The author records a case of a few weeks' standing in which the patient, a European woman in Hong Kong, was otherwise perfectly healthy, showing none of the signs of the pre-pellagrous state. Her diet had been well balanced. She was given 50 mgm. nicotinic acid night and morning by the mouth and within 72 hours the tongue was almost clean.

The aetiology of the condition is not clear and the author states that so far as is known there is no connexion with canine black tongue. This being so, and in the absence of symptoms of pellagra, he is unable to account for the dramatic response to nicotinic acid, which he had not expected to produce any effect.

ii. In this case there was no enlargement of the filiform papillae, but the patient had an abscess round a decayed molar tooth. There were several silver amalgam fillings in the mouth, and as the black tongue cleared up after removal of the diseased molar, the authors think that the black coloration may have been due to interaction between the silver and the septic discharges, resulting possibly in a deposit of silver sulphide on the tongue. The question of avitaminosis is not mentioned in this case.

C. W.

NAGANNA (B.), GIRI (K. V.) & VENKATESAM (P.). **Urinary Excretion of Nicotinic Acid in Pellagrins.**—*Indian Med. Gaz.* 1941. Apr. Vol. 76. No. 4. pp. 208–210. With 2 figs.

This is an article detailing a modification of the method introduced by HARRIS and RAYMOND (1939) for the quantitative estimation of nicotinic acid in the urine.

All methods are based on the reaction in which the pyridine nucleus is broken down by cyanogen bromide and an aromatic amine with the formation of a yellow compound. The aromatic amine has varied in different methods, that used by HARRIS and RAYMOND being p-amino-acetophenone; for estimating the intensity of the yellow colour the Pulfrich photometer was employed. The authors' adaptation of this technique consists in substituting an ordinary colorimeter for the photometer, which is not to be found in every laboratory. The nicotinic acid values determined by this method were 4·8 to 6·1 mgm. in the 24 hours for healthy adults taking an adequate diet, compared with 3·0 to 5·0 mgm. of Harris and Raymond. In pellagrins the amounts found varied from 0·0 to something under 3·0 mgm., normal values being found after successful treatment of the cases with nicotinic acid.

H. S. S.

SYDENSTRICKER (V. P.). **The Present Status of Nicotinic Acid.**—*Arch. Intern. Med.* 1941. Apr. Vol. 67. No. 4. pp. 746–754. [22 refs.]

TROWELL (H. C.). **A Note on Infantile Pellagra.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1941. July 3. Vol. 35. No. 1. pp. 13–20. With 2 plates. [21 refs.]

The author returns to the subject of pellagra in young children and some allied conditions reported from Africa and elsewhere. The article is devoted to a discussion of the differences between his own views and those of Dr. Cicely WILLIAMS [this *Bulletin*, 1934, Vol. 31, p. 344 ; 1940, Vol. 37, p. 798]. Though impossible to summarize it will be read by all those interested in the subject, though many of the points raised have already been dealt with elsewhere. Dr. Trowell believes that these cases should be collected under the heading of pellagra [rightly in the opinion of the reviewer, who some years ago put in a plea to that effect, if only for the benefit of those who have to deal with the literature of the subject. To entitle an article " Proctor-Williams Disease " or " Kwashiorkor " is indefensible. In view of our still very incomplete knowledge concerning disorders due to malnutrition it would be so much wiser, in many cases, if observers would be satisfied with giving clear and detailed descriptions of the many conditions met with in children without attempting to assign to them *precise* aetiological labels ; great care too is needed in choosing descriptive terms, a point well illustrated in Dr. Trowell's article.] In discussing the designations " mosaic skin " and " crazy-pavement skin " he would like to see these terms defined in accordance with his own views and complains that other writers use them in a different sense. [If any differentiation is to be made it should be, the reviewer would suggest, in relation to the skin areas enclosed by the cracks—" mosaic skin " when the areas are small, " crazy pavement skin " when they are large—whereas it would appear that Dr. Trowell uses these terms in the opposite sense. He would apply the term " mosaic skin " to the smooth darkened skin to be noted over the shins having the appearance of cracked varnish, which he asserts desquamates only slightly and never peels in large plaques, an observation which is not quite correct, as under treatment exfoliation in large plaques may occur and be completed very quickly.] *H. S. S.*

MCKENZIE (Alan). **Crazy Pavement Skin Eruption.** [Correspondence.] —*Trans. Roy. Soc. Trop. Med. & Hyg.* 1941. July 3. Vol. 35. No. 1. pp. 47–48.

The writer of this letter examined 568 school children at Morogoro, Tanganyika Territory, and found that crazy pavement skin eruption was not uncommon in conditions far removed from those described by TROWELL (above). In an assessment of nutrition three groups of children were recognized : I, those markedly thin and backward at games, II, those reasonably well nourished and displaying average energy, and III, those above the average in physique and behaviour. It was found that the skin condition was more common in girls than in boys, less common in the older age groups and in children of good physique, but in younger children the incidence was not markedly greater in the obviously poorly nourished than in those of average

physique. It was not associated with any other sign of malnutrition and was rarely found in conjunction with signs of lack of vitamin A. The distribution is shown in the accompanying table.

	Group I	Group II	Group III	Total
Primary boys	34	167	39	240
Percentage with lesion	23·6	21·0	10·3	19·2
Primary girls	13	37	16	66
Percentage with lesion	46·2	40·5	6·2	33·5
Secondary boys	32	152	78	262
Percentage with lesion	31·2	12·5	8·9	13·8

C. W.

TROPICAL OPHTHALMOLOGY.

A Review of Recent Articles. XXXVIII.*

Purulent Ophthalmia.—The successful treatment of gonorrhoeal ophthalmia by the insufflation of a sulphanilamide preparation into the conjunctival sac has been reported by Panneton.[1] Ten cases are reported, seven of them being ophthalmia neonatorum.

Cornea.—An account of an epidemic of *superficial punctate keratitis* in Malaya has been given by Viswalingam.[2] The disease was first observed in the Federated States in 1935 and was present in epidemic form from 1935 to 1938. Its characteristics differed little from those observed in India and elsewhere. The attack usually started as an acute conjunctival inflammation, often accompanied by chemosis, in the region of the outer canthus. The inflammation was commonly associated with swelling and tenderness of the pre-auricular gland. The extent and severity of the corneal lesions seemed to be in direct proportion to the conjunctival inflammation. No herpetic eruptions were observed in any of the patients. The author believes that the disease is caused by an exogenous infection and that the infecting agent is probably a virus.

Encouraging results in the treatment of inflammatory conditions of the cornea by the intravenous injection of ascorbic acid have been reported by Keith Lyle and McLean.[3] They record 10 cases of various forms of keratitis so treated, three of them being superficial punctate

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* For the 37th of this series see Vol. 38, pp. 345–347.

[1] Panneton (Philippe). Local Treatment of Gonorrheal Conjunctivitis with Sulfanilamide Powder.—*Amer. Jl. Ophthalm.* 1941. Mar. Vol. 24. No. 3. pp. 314–321.

[2] Viswalingam (A.). Epidemic Superficial Punctate Keratitis in Malaya.—*Brit. Jl. Ophthalm.* 1941. July. Vol. 25. No. 7. pp. 313–324. With 5 figs.

[3] Lyle (T. Keith) & McLean (D. W.). Vitamin "C" (Ascorbic Acid)—its Therapeutic Value in Inflammatory Conditions of the Cornea.—*Brit. Jl. Ophthalm.* 1941. June. Vol. 25. No. 6. pp. 286–295.

keratitis, and in most the improvement was almost dramatic. The intravenous injections used were of ascorbic acid 'Roche,' a concentrated form of vitamin C in ampoules of 500 mgm. per injection. Ascorbic acid appeared to be useless in the treatment of iritis.

Cataract.—SALIT[4] in a study of the records of 1,863 patients, suffering from senile cataract, who attended the eye clinic of the State University of Iowa during the past 29 years, observed a periodical fluctuation in the number of admissions. A study of the meteorological statistics during these years showed that each rise in incidence of senile cataract immediately followed, or occurred during, a state-wide period of drought. Of the 1,863 patients 1,152 were men, a fact which suggests that exposure to the weather may have been a factor.

GILLETTE[5] has reported his treatment of 20 sufferers from senile cataract by the instillation into the conjunctival sac of a solution of thyroxin twice daily. None of the patients benefited by the treatment.

Retinitis pigmentosa.—As a means of improving the circulation in the retina Max BROWN[6] has used retrobulbar injections of atropine sulphate in the treatment of retinitis pigmentosa. He injected 1 cc. of a 1-1000 solution of the drug twice a week. He states that out of six patients the disease was distinctly improved in two, arrested in three, and unaffected in one.

Lymphogranuloma inguinale.—MACNIE[7] has recorded three cases of uveitis, in the causation of which lymphogranuloma inguinale may have played a part. His paper furnishes a comprehensive review of the subject and he concludes that the virus of lymphogranuloma is one of the causes of Parinaud's syndrome and may be a cause of other ocular diseases, particularly those of the uveal tract.

Rhinosporidium seeberi.—A case of rhinosporidial infection of the conjunctiva occurring in Texas has been reported by ELLES.[8] The patient was a boy aged 14 and the growth was attached to the upper tarsal conjunctiva. The polypus had the typical appearance—leaf-like, red, and studded with small yellowish specks. "The whole mass had the appearance of a lily pad." The growth was freely excised and there has been no recurrence in a period of two years. The author gives a full review of the literature and refers to two previous cases observed in the U.S.A., one in North Carolina and the other in Southern Texas.

H. Kirkpatrick.

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[4] SALIT (P. W.). Droughts as a Factor in the Development of Senile Cataract.—*Amer. Jl. Ophthalm.* 1941. Mar. Vol. 24. No. 3. pp. 310–314.

[5] GILLETTE (David F.). Effect of Thyroxin on Incipient Senile Cataracts.—*Arch. Ophthalm.* 1941. Mar. Vol. 25. No. 3. pp. 438–442. [20 refs.]

[6] BROWN (W. Max). Treatment of Retinitis Pigmentosa with Retrobulbar Injections of Atropine Sulfate.—*Arch. Ophthalm.* 1941. May. Vol. 25. No. 5. pp. 819–826. With 6 charts.

[7] MACNIE (John P.). Ocular Lymphogranuloma Venereum.—*Arch. Ophthalm.* 1941. Feb. Vol. 25. No. 2. pp. 255–279. With 6 figs. [54 refs.]

[8] ELLES (Norma B.). Rhinosporidium Seeberi Infection in the Eye.—*Arch. Ophthalm.* 1941. June. Vol. 25. No. 6. pp. 969–991. With 6 figs. (1 coloured). [48 refs.]

MISCELLANEOUS.

JOLLY (G. G.). **Rural Medical Relief.**—*Indian Med. Gaz.* 1941. Mar. Vol. 76. No. 3. pp. 177–180.

[This paper is primarily concerned with India, but it may be that the system proposed, if successful there, could be applied to other countries.] The author points out that although hospitals are usually situated in towns, they cater for a considerable number of patients from rural areas, but in spite of this the great mass of villages in India have grossly inadequate arrangements for medical relief. Rural medical relief can only be founded on resident medical practitioners, but in view of the poor returns obtained and the lack of amenities in the villages, it is not surprising that practitioners prefer to remain in towns. The solution appears to be Government subsidy to practitioners working in villages; such a scheme is working successfully in Madras and has been started in the Punjab. Rural dispensaries constitute another problem; they should act as cottage hospitals and should be so organized that they encourage the settlement of rural practitioners by acting as a link between them and the larger hospitals. These cottage hospitals should serve village groups, acting as the counterparts in civil life of the casualty clearing stations of war. *C. W.*

LEDGER (L. K.) & WASSON (Tek Chand). **Ages of Epiphysial Union at Elbow and Wrist Joints amongst 238 Children in North West Frontier Province.**—*Indian Med. Gaz.* 1941. Feb. Vol. 76. No. 2. pp. 81–84.

This work is a continuation of that done by LALL and TOWNSEND and reviewed in this *Bulletin*, 1940, Vol. 37, p. 384. The age records of 189 boys and 49 girls were checked and confirmed, and X-ray examination was then made on these subjects. The full results are set out in a series of tables which cannot be reproduced here, and the authors admit that the series is small and that the margin of error must be high. There are great differences in the ages at which ossification of the same epiphysial lines take place. So far as these results go, however, they indicate that epiphysial union takes place earlier in India than in Europe, and earlier in girls than in boys. The ages at which teeth erupt vary widely; girls appear to get their wisdom teeth later than boys, though the epiphyses unite earlier. By the age of 14, 54 per cent. of the girls had started to menstruate, by the age of 15 this had risen to 75 per cent.

An opinion as to age in adolescence cannot, therefore, be given with great accuracy, and for medico-legal purposes there must be taken into account—the union of six epiphyses (radius, upper and lower; ulna lower; humerus, lateral and medial condyles; base of the first metacarpal), the number of erupted teeth, and the date of menstruation. Even then, however, a margin of about six months each way should be allowed. *C. W.*

BROUWER (D.). De bezinkingssnelheid der erythrocyten en haar beteekenis in de tropen. [**The Significance of the Blood Sedimentation Rate in the Tropics.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1940. Sept. 10. Vol. 80. No. 37. pp. 2184–2202. [21 refs.] English summary.

" After a discussion of the technique and the influence of the laboratory-temperature on the sedimentation rate of the red blood corpuscles, the author gives a short review about the theory of the sedimentation.

" At Batavia-C., working with a laboratory temperature of 25 degr. C., he found as normal rates 4–11 mm. in the first hour in young, healthy men, Europeans, Eurasians, Chinese as well as natives who lived under good health- and food-conditions.

" This is in correspondence with the researches on the proteins of the bloodplasma STREEF and STREEF-SPAAN made in young healthy pupils of the medical school at Sourabaja, mostly Chinese and natives.

" They found protein levels corresponding with those from Europe.

" In the tropics very often among the poor classes of the population a rather high increase of the sedimentation rate of the red blood corpuscles can be found. The causes are discussed : *underfeeding*, *beri-beri* and *ankylostomiasis*. Author found in a large number of patients a decrease of the sedimentation rate of the red blood corpuscles of 3 mm. and therebelow in the first hour.

" He thinks there is a causal relation with a *more or less latent amoebiasis of the liver*.

" Patients come with different complaints. From a clinical point of view, one can divide them in different groups :

" A. Asthenic complaints. Fatigue, not feeling fit and such like.

" B. Indefinite stomach complaints.

" C. Pseudo-biliary colics.

" D. Allergic symptoms. (Asthma, migraine, urticaria).

" With all these patients the liver was found enlarged. They all had in their clinical history, or still had, an amoebiasis of the intestines. The intestinal symptoms can be so slight that dysentery has never been complained of.

" Author thinks that the penetration of the amoebae in the liver causes a sort of toxic edema (hepatitis serosa), which causes an analogy with cases of icterus catarrhalis, a decreased sedimentation rate of the red blood corpuscles. All cases reacted well to emetine hydrochloride. In many patients the sedimentation rate could be followed for a longer period.

" In mild cases where the liver was sensitive on pressure but not enlarged, the sediment rate after treatment with emetine injections became normal in about six weeks. When the liver was already enlarged, the sluggishness of sedimentation was less inclined to disappear and in most cases the course of emetine injections was repeated after six weeks.

" The sedimentation rate of the red blood corpuscles proved to be a valuable diagnostic help in tropical practice."

[See also this *Bulletin*, 1940, Vol. 37, p. 811.]

FARNESS (O. J.). **Coccidioidomycosis.**—*Jl. Amer. Med. Assoc.* 1941. Apr. 19. Vol. 116. No. 16. pp. 1749–1752. With 6 figs. [16 refs.]

Hitherto most of the cases of coccidioidomycosis have been reported from California—San Joaquin Valley fever is one of its synonyms—but the present article shows that it occurs outside that State, the series described here having been reported from Arizona.

The author gives a description of the fungus, *Coccidioides immitis*, showing that it occurs in two forms or cycles, one, the parasitic, found in infected tissue ; the other, the vegetative, in culture and probably in nature. The former, in infected tissue and sputum, appears as a spherule, 5–60μ in diameter, with refractile, double-contoured capsule.

This form does not seem to be transmissible, as infection from man to man or animal to man is not known to occur. The portal of entry is usually the respiratory passages, rarely a traumatic cutaneous lesion, and dissemination takes place by blood or lymph stream, or by direct extension. Any organ may be affected, most commonly the lungs, skin, bones, lymph nodes and meninges. Its clinical manifestations are varied and its resemblance in some cases to tuberculosis is such that diagnosis can be made only by finding the characteristic spherules and confirming them by culture and animal inoculation. The same symptoms of chills, cough, blood stained sputum, fever, night-sweats, loss of weight, asthenia, malaise are common to both. An erythematous rash like erythema nodosum may be seen, disappearing in 7–10 days. Death may occur in the acute infection, or dissemination may give rise to a chronic, progressive disease. A specific cutaneous reaction may be obtained with coccidioidin.

The author gives clinical details of five cases: (1) a man of 67 with signs and symptoms of acute pneumonia; (2) a man of 46 with the chronic progressive granuloma form; (3) a boy of 15 with rapid cavitation and strong resemblance to tuberculosis (this patient died); (4) another lad of 15, with the febrile, broncho-pneumonic, San Joaquin Valley fever form; (5) a woman of 23 with a very acute form following full-term parturition: death occurred 19 days after onset of the illness with symptoms like those of acute miliary tuberculosis [see this *Bulletin*, 1938, Vol. 35, p. 459; 1939, Vol. 36, pp. 101, 507, 508; 1940, Vol. 37, p. 882]. *H. H. S.*

KESSEL (John F.). **Recent Observations on Coccidioides Infection.**—*Amer. Jl. Trop. Med.* 1941. May. Vol. 21. No. 3. pp. 447–453. [15 refs.]

An excellent account of the subject, largely from the epidemiological aspect. Between 1893 and 1939 there were 660 cases of coccidioides granuloma reported to the California State Department of Public Health. The following table shows the rapid increase in more recent years:—

Coccidioidal Granuloma in California.

1893–1899	4
1900–1904	7
1905–1909	9
1910–1914	17
1915–1919	23
1920–1924	50
1925–1929	123
1930–1934	163
1935–1939	264
Total	660

Most of these originated in the southern part of the San Joaquin Valley and of 100 patients admitted to the Los Angeles County Hospital, 51 acquired the infection in San Joaquin Valley, 31 in California south of Tehachapi, eight in Arizona, New Mexico or Texas; 10 were not determined.

The chlamydospores are in the dust and the commonest portal of entry is the respiratory tract, by inhalation of the spore-laden dust;

in some 70 per cent. the initial lesion is in the lung. Next commonest is an abraded site on the skin. A typical instance of this is described. A man went hunting near Lake Elsinore, Riverside County. His leg was abraded by the top of his boot and the lesion failed to heal. He ran a temperature indicative of sepsis, blood culture yielded *Coccidioides immitis*, and the lungs showed slight involvement. He died in a fortnight from miliary coccidioidosis.

The chronic " granulomatous " form may involve any organ and the death rate is 50–60 per cent. Pulmonary disease is most common; generalization occurred in 16 of 50 cases studied. With generalization meningitis is usual and death soon ends the scene.

Other patients suffer from respiratory symptoms commonly diagnosed as a cold, or influenza, or bronchopneumonia, and recovery is usually fairly prompt. In about 5 per cent. a rash like that of erythema nodosum is seen ; the death rate is under 1 per cent. These infections are termed San Joaquin Valley fever, Desert fever, or Desert rheumatism. The coccidioidin test with a preparation made of a mixture of several strains appears to be very specific, and recovered cases have been known to react as long as nine years after the initial infection.

H. H. S.

FOLEY (Maurice P.), LOVE (J. Grafton), BRODERS (Albert C.) & HEILMAN (Fordyce R.). **Coccidioidal Granuloma : Report of Case Originating in Texas.**—*Western Jl. Surgery, Obstet. & Gynecol.* 1940. Dec. Vol. 48. No. 12. pp. 738–741. With 5 figs. [15 refs.]

Coccidioidal granuloma can no longer be regarded as a rarity. More than 450 cases have now been reported from California ; most have developed in San Joaquin Valley but it would be wrong to infer that it is not found elsewhere. It is probable also that deaths occur from it, the condition having been diagnosed as tuberculosis.

The authors record a case in an office worker, 33 years of age, from north-western Texas. He complained of fever, chronic cough, night sweats, fatigue, headache, transient blurring of vision and a discharging sinus behind the right ear. He had been ill for about 10 years on and off (since 1927) and had lost 43 lb. in weight. Two years ago there developed two small tender swellings the size of a pea, over the right occipital region ; aspiration the following year yielded material containing spore-like bodies, and culture a fungus-like growth, thought to be a streptothrix.

Examination in 1937 showed leucocytosis of 11,100 per cmm. of blood, W.R. negative, no tubercle bacilli in the sputum ; X-ray showed three destructive lesions of the occipital bone, and lesions in the lungs simulating active tuberculosis. Operation revealed a mass of shiny granular tissue, pearly in appearance extending through the skull and over the dura. It was carefully scraped off and the site swabbed with Tr. iodi. Convalescence was rapid. Cultivation and animal inoculation confirmed *Coccidioides immitis* as the cause.

The authors describe the organism and its lesions in the following words :—

" Coccidioides immitis, a doubly contoured organism frequently filled with spores, usually gains entrance to the body by way of the skin or lungs. It attacks the skin, lungs, lymph nodes, bones and so forth, with an avidity comparable with that of Mycobacterium tuberculosis. It produces nodules, ulcers, tubercles and abscesses and presents a macroscopic

picture for the most part comparable with that of tuberculosis. With the formation of tubercles consisting of epithelioid cells, foreign body giant cells and necrosis, the microscopic picture is even more like tuberculosis than the macroscopic—and if one did not find the double contoured organisms, often containing spores, one would be justified in making a diagnosis of tuberculosis."

In its growth it presents two phases : a round unicellular body with refractile double contoured border and containing endospores ; this form is seen in animal inoculations. The other shows branched, septate mycelia, without conidia, this is seen in cultures on artificial media. [See this *Bulletin*, 1937, Vol. 34, pp. 177, 510 ; 1938, Vol. 35, p. 459 ; 1939, Vol. 36, pp. 507–508 ; 1940, Vol. 37, p. 882 ; 1941, Vol. 38, p. 94.] *H. H. S.*

Niño (Flavio L.), Pons (Luis M.) & Gay (Antonio E.). Granuloma paracoccidioídico de localización laríngea. (Estudio clínico y micológico.) [**Laryngeal Paracoccidioidal Granuloma.**]—*Bol. Inst. Clín. Quirúrg.* Buenos Aires. 1941. Jan. Vol. 16. No. 138. pp. 47–65. With 16 figs.

The patient was an Italian, 30 years old, who had had a " cold " for four months, a sense of heat in the nasal mucosa, and for 2–3 weeks, some difficulty and pain on swallowing ; there was no fever. On the left side of the neck were small, hard, painless lumps the size of peas and a gland as large as a filbert. The uvula was oedematous and at the base on the left side there was a small ulcer, 5 mm. in diameter, and behind it another 1·5 cm. in diameter with yellowish white slough and swollen edges. The left side of the epiglottis was oedematous and ulcerated and these conditions extended to the larynx.

He was given radium treatment, with iodide internally up to 6 gm. daily. The ulcerations improved and the general condition remained good. By growth from the expectoration and by animal inoculation of fragments taken for biopsy *Paracoccidioides brasiliensis* var. *cerebriformis* was proved. This strain, at all events, has not proved very virulent, as the condition has now been present for two years since symptoms appeared and the patient's health remains generally good.

H. H. S.

Dormer (B. A.) & Friedlander (J.). **Case Report from the Clinical Department of King George V. Jubilee Hospital for Tuberculosis, Durban.**—*Brit. Jl. Tuberculosis.* 1941. Apr. Vol. 35. No. 2. pp. 114–115. With 3 figs. on 2 plates.

Unfortunately the case here reported could not be followed to its conclusion, but is worth notice because of its resemblance to one of infection by *Coccidioides immitis* and of which it may possibly have been an example.

A native woman of 33 years of age, developed a dry, non-productive cough which after a month became looser, with increasing mucopurulent sputum. She had no night-sweats, but on one occasion a slight haemoptysis. There was progressive dyspnoea. When she came under the authors' observation, there was marked orthopnoea, cough, profuse mucopurulent, nummular sputum, no adenitis, no noticeable emaciation. The physical signs were those of fairly advanced bilateral pulmonary tuberculosis. No *Myco. tuberculosis* could be seen ; moulds and secondary organisms grew on culture and "round bodies" were

seen. Red blood corpuscles numbered 5,130,000, leucocytes 13,500 per cmm. X-ray appearances were those of miliary tuberculosis with pneumonic consolidation of the right upper lobe and radiologically and clinically the patient presented the indications of a rapidly advancing tuberculosis. She left hospital and died outside; no post-mortem examination could be made. *H. H. S.*

THORNER (Juliet E.). **Coccidiomycosis. Relative Values of Coccidioidin and Tuberculin Testing among Children of the San Joaquin Valley.**—*California Western Med.* 1941. Vol. 54. No. 1. pp. 12–15. With 2 graphs. [Summarized in *Rev. Applied Mycology.* 1941. May. Vol. 20. Pt. 5. p. 203.]

"Tests were conducted simultaneously with 0·1 cc. of Koch's O.T. 1 : 1,000 and coccidioidin from *Oidium coccidioides* [*Coccidioides immitis*] on 267 children selected at random from the Pediatric Clinic of the Kern General Hospital, and ranging in age from 5 months to 19 years. There was found to be a noticeably higher incidence of coccidioidin over tuberculin reactors as the period of residence in Kern County increased, amounting to 76 over 25 per cent. after ten years. Among a group of 42 patients with markedly positive skin reactions to the coccidioidin the stomach washings of only two proved positive for the fungus on culture and guinea-pig inoculation. The lower ratio of 'valley fever-coccidioidin' reactors observed in this and other surveys points to the relatively benign as well as universal nature of the primary phase of coccidioidomycosis as it exists in the San Joaquin Valley. Progression into the later and severe granulomatous stage of the disease only takes place when tissue and lymphatic resistance are overcome and generalized dissemination is effected through the blood stream."

BLANCO (Manuel C.). Las micosis broncopulmonares. Su importancia clínica. [**Bronchopulmonary Mycoses.**]—*Bol. Inst. Clín. Quirúrg.* Buenos Aires. 1940. Dec. Vol. 16. No. 137. pp. 787–930. With 41 figs. [223 refs.]

This is a carefully compiled monograph on an important subject; it is well illustrated and fairly fully documented. The work is divided into thirteen chapters and a bibliography or list of over 200 references culled from many sources, British, American, Spanish, French, and even some Italian.

The first chapter deals with the history of mycoses from the studies of MALPIGHI in the latter part of the seventeenth century. The second treats of the morphology, biology, reproduction, and classification. The author's classification of them is into three main groups: I. Fungi with blastospores, with two families Saccharomycetaceae and Torulopsidaceae. II. Fungi with astrospores. III. Filamentous fungi—Actinomycetaceae with thin filaments, Mucoraceae and Aspergillaceae with thick filaments. This section is excellently illustrated. The next two chapters treat of the pathogeny and pathological anatomy, rather briefly but fairly adequately, the morbid histology being illustrated by schemes of the lesions and photomicrographs. Symptomatology is sketched, the chief symptoms being cough, expectoration, haemoptysis (which is common), thoracic pain, wasting and fever. Laboratory diagnosis rests on examination of the sputum and the results of culture, together with intradermo-reactions, complement fixation tests and animal inoculation. Differential diagnosis (Chapter VII) has to be made from tuberculosis, syphilis of the lung, cancer, abscess, hydatid

and other helminthic infections, bronchospirochaetosis, and pulmonary gangrene. Succeeding chapters deal with clinical forms, associated diseases, treatment (mainly by potassium iodide), prophylaxis, evolution and prognosis. Next come clinical accounts of 23 patients, and a summing up of the author's conclusions.

Pulmonary mycosis is thought to be rare and medical men report cases from time to time as if they were curiosities, but it is not a very rare disease, certainly in the Argentine, and reports of cases are increasing in numbers. Some of the fungi responsible are true parasites, others are "pseudoparasites"; the latter may acquire pathogenicity when the local conditions in the mucosa or character of the secretions favour the change. The former attack the lungs and produce their effects, partly mechanically owing to their situation, partly as the results of the toxins they produce, with local inflammatory reaction. The clinical diagnosis must be confirmed by laboratory investigation. Prognosis is, on the whole, favourable, provided the condition is recognized early and treated thoroughly; otherwise local spread and dissemination occur with formation of abscesses and sinuses, invasion of the pleura and thoracic wall and perhaps septicaemic generalization. The prognosis in an established case depends, therefore, on the clinical form, the nature of the infection, the extent of the lesions, the general state of the patient and his ability to tolerate intensive iodide treatment.

H. H. S.

AIRES (Octavio). Um caso de aspergilose pulmonar. [**A Case of Pulmonary Aspergillosis.**]—*Folha Med.* 1941. Mar. 25. Vol. 22. No. 6. pp. 66–68.

The patient was a woman of 75 years who stated that two months before coming under the author's care she had an attack of "influenza," not severe enough, however, to force her to lie up. This was soon followed by a cough and expectoration. A few days later she felt feverish, suffered from headache and noticed that she was getting thinner.

On examination she was observed to be emaciated, very weak and needed assistance to rise in bed. Physical examination of the chest showed little abnormal, though X-rays indicated a dense area at the left base. There was copious greenish-yellow, mucopurulent fetid expectoration, in which no tubercle bacilli could be seen, but *Aspergillus fumigatus* was cultivated on Sabouraud's medium, and mycelial filaments and spores could be seen in stained smears. This fungus is not an uncommon infection in birds, and man may be infected by close association with caged birds; it also, of course, is found in grasses and contaminating rice, millet, and rye. In the present instance the source was not determined. *H. H. S.*

MAZZOTTI (Luis). Existencia de la pseudolepra de Roblès en México. [**Pseudo-Leprosy of Roblès in Mexico.**]—*Medicina.* Mexico. 1941. Mar. 10. Vol. 21. No. 383. pp. 103–107. With 3 figs.

Fourteen years ago Dr. Rodolfo ROBLÈS described under the name of *punudos* a condition which he had seen in Guatemala, but was

unable to classify. He called it also " pseudo-leprosy " [the reason of this is obscure]. It is an elephantiasic condition of the feet, occurs in either sex, usually begins in childhood as an erythema of the instep with adenitis, some fever and malaise. After 4–5 days the redness subsides leaving some oedema ; recurrences take place at varying but usually progressively shorter intervals, till the whole foot (in time both feet) is swollen and papillomata develop on the dorsum and digits. Local infection among a people going about barefoot is likely, but no parasites or causative bacteria have been found, and heredity does not seem to play a part [see this *Bulletin*, 1928, Vol. 25, p. 703.]

H. H. S.

SIMONS (R. D. G. Ph.). Apenpokken en roode hond. [**Pemphigus Contagiosus and Prickly Heat.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1941. Jan. 13. Vol. 81. No. 2. pp. 59–86. With 4 figs. on 1 plate & 1 folding plate. [15 refs.] English summary.

" Detailed review of the ' monkey-pocks ' (the name of pyosis mansoni or pemphigus contagiosus in the Netherlands East Indies) and prickly heat : Staphylodermia bullosa superficialis non-follicularis *disseminata* endemica (epidemica) tropica and miliariasis *erythemato*-papulosa tropica.

" European and Indo-European people are more prone to both diseases than native people, who are very rarely affected.[1]

" Because the staphylococci prefer the folliculae, sweat- and sebaceous glands, it is of importance to note, that this is not the case with pemphigus contagiosus. The bullae are localised *inter*-follicularly.[2] The pustule formation only occupies the epidermis. The bullae do not leave scars. The not-itching disease is contagious. Should no general hygienic measures be taken the disease continues to exist in the hospital.

" Pemphigus contagiosus tropicus and pemphigus neonatorum are identical.[3] Pyosis Corletti and pyosis Mansoni are probably the same as these ' monkey-pocks '.

" Prickly heat is very often mistaken for seborrhoeic eczema. The name lichen tropicus is wrong or will not meet the case. The cause is obstructed perspiration, or better : the obstructed evaporation of sweat in climates with a high percentage of moisture of the air. The skin is irritated by perspiration (also by alteration of the pH) and infected. Microscopically there is a non-humid eczema plus a pyodermia. Should the prickly heat become humid one may assume that there has been an irritation by medicaments, e.g. soaps. The region of the eccrine sweat glands is specially affected. Greasy ointments are not to be recommended for both diseases. The disinfection treatment of the monkey-pocks is very useful and of diagnostic value.[4] This treatment consists of frequent bathing, disinfection of the clothes, and should be continued to prevent relapses. For prickly heat lotions are preferable to soaps, which sometimes may provoke the prickly heat or irritate the (eczematous) skin. In the tropics after every bathing dusting powders should be freely applied to the normal skin and skin affected by prickly heat. By this *radiator*-treatment the (perspiration) surface of the skin is enlarged and all moisture is absorbed. All additional causes (e.g. alcoholic drinks, hot fluids and food, warm clothing, elastic parts of the clothes, soft sleeping mattresses) of perspiration must be avoided."

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[1], [2], [3] and [4] are the characteristic peculiarities of pemphigus contagiosus.

HOWELL (D. E.) & STILES (George W.). **An Unusual Reaction to the Bite of the Dove Louse Fly,** *Stilbometopa podopostyla* **Speiser.**—*Jl. Amer. Med. Assoc.* 1941. Apr. 5. Vol. 116. No. 14. pp. 1517–1518.

There are few records of man being bitten by hippoboscid flies, and in general the effects of the bites are slight. The patient here recorded was bitten in the region of the submaxillary gland by the dove lousefly *Stilbometopa podopostyla*, the fly being captured and identified. The bite was not painful, but 2½ hours afterwards the patient suddenly found her vision blurred and lost the use of her arms and legs ; vertigo was extreme and her head ached severely. There was slight local swelling and considerable tenderness of the gland ; the temperature was 97·5°F., the pulse 100. Fleeting pains were felt in the joints, with abdominal soreness, increased respiration, tremors and chilly sensations ; there was no vomiting. Opiates were given, but the symptoms persisted for 28 hours ; complete recovery took place within a few days.

Stilbometopa podopostyla is commonly a parasite of doves and has not previously been known to bite man ; it cannot live away from its host for more than a few days and presumably in this instance there were doves near the house, though none had been seen. *C. W.*

GALLIARD (Henri) & DANG-VAN-NGU. Une espèce nouvelle d'*Anophèles* du Tonkin, *A. tonkinensis* sp. nov. [*A. tonkinensis* **a New Species from Tonking.**]—*Rev. Méd. Française d'Extrême-Orient.* 1940. Nov.–Dec. No. 9–10. pp. 595–598. With 3 figs.

TOUMANOFF (C.). Contribution à la connaissance des Ixodidés de l'Indochine du gen. *Haemaphysalis* Koch. C. L. Description de trois espèces nouvelles. [**Haemaphysalis Ticks in Indo-China : Three New Species.**]—*Rev. Méd. Française d'Extrême-Orient.* 1940. Oct. No. 8. pp. 463–490. With 17 figs.

JÍROVEC (Otto) & RODOVÁ (Helena). Ueber die Züchtung der Trichomonaden. [**Trichomonad Culture.**]—*Zent. f. Bakt.* I. Abt. Orig. 1940. Apr. 2. Vol. 145. No. 6. pp. 351–360. [23 refs.]

After considerable experimentation, the authors have devised a medium, for the growth of trichomonads, which is not only simpler than others but better in its reliability. It is prepared as follows :—5 cc. of human or horse serum, which need not be sterile, is diluted with 95 cc. of a buffered modification of Ringer's solution (distilled water 1,000, NaCl 6, $CaCl_2$ 0·1, $MgSO_4$ 0·1, KH_2PO_4 3, n/l NaOH 18–22 to a *p*H of 7·4). The diluted serum is distributed in test tubes, 4–6 cc. in each, and the tubes are then sterilized on two or three successive days in the steam sterilizer. The tubes, which can be kept indefinitely in the ice chest, are then ready for use. In place of the Ringer's solution 0·6 per cent. sodium chloride solution may be used for diluting the serum. In this case the required *p*H of 7·0–7·4 is obtained by the use of n/50 HCl or n/50 NaOH, as the case may be. In this medium a number of human trichomonad strains from the mouth, vagina and intestine, and others from the rat, pigeon and frog have been successfully cultivated, the intestinal ones at room temperature, the others at 37°C. The maximum growth occurs on the fourth or fifth day and subculture

has to be made every seventh day. By covering the medium with a layer of paraffin these times are considerably extended. The trichomonas of the frog will continue to grow in the medium for two months. The advantage of the medium resides in the fact that at the serum dilution employed there is no coagulation followed by deposit, but merely a clouding of the whole liquid, which has no tendency to separation of a coagulum. *C. M. Wenyon.*

REVIEWS AND NOTICES.

HENRIKSEN (Sverre Dick) & OEDING (Per). **Medical Survey of Tristan da Cunha. Results of the Norwegian Scientific Expedition to Tristan da Cunha 1937–1938.** No. 5. 147 pp. with 42 figs. [28 refs.] Published by Det Norske Videnskaps-Akademi I Oslo. 1940. I Kommisjon Hos Jacob Dybwad. [Kr. 8.50.]

A Norwegian scientific expedition visited Tristan da Cunha and the neighbouring islands of Nightingale and Inaccessible during the period December 7th, 1937, to March 29th, 1938. The survey under review is the result of the investigations of its medical members. Their object was to collect information regarding the medical history of the community, their physical characteristics, the bacterial flora of mouth, nose and throat, blood characteristics, Schick, Dick and syphilitic reactions, and to make a hygienic survey of the colony as regards nutrition, housing, and conditions and habits of life.

The settlement was established in 1816. Its present population of 188 individuals is descended from 15 ancestors, 11 of whom were white (Scotch, English, Irish, American, Italian, and Dutch) and four coloured (of mixed negroid origin). The degree of inbreeding is thus considerable. Contact with the external world has, of late years, decreased greatly, and the Tristanites are at present short of nearly everything except what they can themselves produce or gather. Conditions of life are rather primitive, the people are conservative and lack initiative. Latterly the fertility of marriages has been decreasing, as has also the standard of living. The staple diet has always been fish and potatoes, and to a moderate extent milk, meat, seabirds and their eggs. The poorer families eat more animal food than the richer. Vitamin C seems to be in sufficient quantity in the diet of the richer. No statement can be made regarding the other vitamins, except that those parts of fish which contain most of the fat soluble vitamins are not eaten. The liver is invariably thrown away. Potato crops often fail, but " Tristanites are obviously used to hardships and they submit to bad years and starvation with a remarkable equanimity."

All reports seem to show that in spite of a monotonous and often insufficient diet, and of a rough and wet climate, the conditions of health have been very good. Many diseases, frequent in other countries, are lacking in Tristan. With regard to physical development it appears that, whilst adults of both sexes are of about the same height and weight as Norwegians, the children do not come up to Norwegian standards. The older children seem to correspond to Norwegian children two or three years their junior. Thus " the development of Tristan children (of both richer and poorer families) is remarkably slow, and

their state of nutrition is by no means the optimum." The standard of living has definitely deteriorated, imported foodstuffs are increasingly difficult to obtain, the stock of cattle is less, seabirds are diminishing in number and, to prevent their extinction, their eggs are no longer collected; the supply of milk is small. Only the richer families have a diet of reasonably high caloric value, and most never have a supply of food which approaches the usual standards. The calcium and phosphorus content is deficient. These factors may possibly explain the slow development of the children.

This slow development is, however, the only indication that the mode of living of the Tristanites is unsatisfactory. It is not accompanied by ill health. "Otherwise the Tristanites are a very healthy group of people." They are lean and hardy. Deficiency diseases such as rickets, scurvy, hemeralopia, and xerophthalmia are non-existent. No cases of gastro-intestinal affection, except acute dysentery, occur. "Apart from mild dysentery, and an epidemic of colds after each visit of a ship there was no evidence of the presence of infectious disease." Tuberculosis, venereal disease, and acute infections are unknown, and no signs of diseases of heart, joints or kidneys were observed. There was record of two epidemics of mumps, one of jaundice one of sore throat, one of ophthalmia and one of a contagious rash "showing that the Tristanites were not generally immune to infectious diseases." This freedom from infectious diseases is in the opinion of the authors due to the isolated position of the island, which reduces importation to a minimum. Bacterial examination of throat and nose revealed an almost total absence of pathogenic organisms. The only pathogens found were one strain of *H. influenzae*, and a few strains of *Staph. aureus* (probably non-pathogenic). No evidence was obtained of the existence of any endocrine disorders. Blood pressures were, in the majority of persons, normal. In all but a few persons tonsils and adenoids were normal.

The diseases from which the inhabitants suffer fall into three groups: (1) Bacterial infections comprising the common colds and dysentery already referred to. (2) Animal parasites, infection with *Ascaris lumbricoides* being very common, and evidenced also by an almost universal eosinophilia. (3) Hereditary disposition: this is by far the most important group. Hereditary deformity of the external ear accompanied by deafness, was imported from an American whaler in the year 1836, and could be followed through five generations. The most common diseases belong, however, to the allergic group, and these occur with a very high frequency in certain families. Charts showing the inheritance of asthma, migraine and hay fever are given in the text.

Causes of death are classified as under :—

	Male	Female
Accidents	21	2
Inborn weakness	7	2
Old age	2	6
Disease	7	7
Unknown	4	2
Totals	41	19

Among the factors which might be responsible for the good medical record of the island are "the isolated position which keeps infection

away ; the regular and monotonous life ; the fresh and moist climate ; the restricted diet ; the monotony of the diet ; and the composition of the latter, which consists of fresh natural products only. It may be sufficient to state that a mode of living like that on Tristan (or by preference a little more luxurious) seems to have a favourable influence on the health."

The above is a brief summary of this fascinating study, and of the views of those who have made it. Isolation, monotony, hardship even to the verge of starvation, these are strange factors to be associated with good health and well being. Of late years we have heard more often about under-nutrition and the need for a higher standard of living. We did not know that we were undernourished but we are told that we are. We did not realize that a rise in the standard of living, with which we had been content, would benefit us. This refreshing paper sheds a ray of light and hope through the gloom of disillusion and discontent which has been cast over us. That a degree of hardship is a good and sound medicine no one can doubt. Our forebears survived on it—a bag of meal or a dish of brose—and they left a hardy race behind them. Perhaps they were not so misguided after all. Richer diet, comfort and ease are dangerous expedients, as the Romans found to their cost. "Per aspera ad astra." The little island of Tristan da Cunha has told its story. We would do well to pause, and to re-examine the standards which we have set up. Even our enemies must admit that we are a hardy race, and we must see to it that we remain so.

A. G. McKendrick.

WELCH (Janet) [M.B.E., M.B., B.S.(London), M.R.C.S., L.R.C.P. (England), D.T.M. & H.(England), Medical Officer, Church of Scotland Hospital, Blantyre, Nyasaland]. **Nursing Education related to the Cultural Background in East and Southeast African Colonies.**—87 mimeographed pp. 1941. New York: The Carnegie Corporation.

The author of this interesting and valuable report has been engaged for 12 years in medical practice in Kenya and Nyasaland, and recently, by means of a grant from the Carnegie Corporation, has made a special study of modern developments in nursing education, organization and practice, and has visited nursing institutions in Canada, the U.S.A. and Porto Rico.

Modern developments in nursing education in many parts of the world are described, and extracts from numerous reports quoted. One extract from the 1937 report of the International Health Division of the Rockefeller Foundation sums up the position. The Foundation "seeks to further certain modern principles of nursing education, namely, that the responsibility of a school of nursing should be the education of nurses on a professional level, that the School should have its own budget separate from other budgets of the institution with which it is connected, that nursing education should have a broad cultural, social, and scientific basis, that public health nursing should be taught throughout the nursing course, and that it is as important to provide good field practice in public health nursing for the students as it is to teach good bedside nursing care in a hospital."

Part I of the report under review describes the lack of nursing services in East Africa to-day and the difficulties associated with their development, one of the chief of which is the inadequate education of women

and girls. There follows a section on the type of nursing service which is considered necessary. It is felt that the great need in African society is for instruction in preventive medicine, that the best means of giving this instruction is by means of public health or "community" nurses, and that in planning a curriculum for nursing education the preventive and social aspects of the subject should therefore be stressed throughout the whole course.

The English system of training public health nurses, that is, hospital training followed by post-graduate public health training, is not considered suitable for Africa.

Part II deals with the system of nursing education which is thought suitable for East and South-East Africa, and a curriculum is drawn up for training girls who have had a primary school education. It is considered that the nursing schools should be organized as educational institutions and not as service departments to hospitals, and that there should be full-time instructors who would also give bedside instruction in the wards. It is recommended that all European nurses and not only instructors should have preparatory courses in London to equip them to meet the special problems of the African social background.

The author states "The solution of the problem appears to be along the following lines—(*a*) The nursing school must be an educational institute for the undergraduate nurse throughout her course. (*b*) The hospital must be the portal to a planned continued educational process for the student throughout her course. (*c*) The hospital must supply its nursing staff to cover the nursing requirements of all its patients and not be dependent on student service from the school; although this African staff would not be highly trained until graduates under the new educational system were available to take their place. (*d*) Provision must be made for training and employment of such workers as nurse-aids and ward-maids to relieve qualified African staff of non-nursing duties throughout the hospital."

It is suggested that there should be a nursing school in each Colony with its own Administrative Committee and that there should also be a Central Advisory Council to co-ordinate the work throughout all the East and South-East African Colonies. Co-operation between Education and Health Departments both in nursing education and in giving health instruction to the people is urged.

The report should prove very helpful to all those who are concerned in building up nursing services in the colonies, and the development of an experimental nursing school in keeping with modern ideas of nursing education on the lines suggested by the author should be considered. *M. G. Blacklock.*

EDWARDS (F. W.) [M.A., Sc.D., F.R.S., etc.]. **Mosquitoes of the Ethiopian Region. III.—Culicine Adults and Pupae.**—pp. viii+499. With 4 coloured plates & 184 figs. 1941. London: Printed by Order of the Trustees of the British Museum. [£1 10*s*.]

This volume, which deals with the adult Culicine mosquitoes and their pupae, completes the monograph of the mosquitoes of the Ethiopian Region, that is, the whole continent of Africa south of the Sahara. Part I by G. H. E. HOPKINS [this *Bulletin*, 1936, Vol. 33, p. 570] covered the larvae of the Culicines and Part II by

A. M. EVANS [this *Bulletin*, 1938, Vol. 35, p. 930] the adults and early stages of Anophelines. The Culicines comprise about 290 species and some 40 named subspecies and varieties. Many of these are now described for the first time: all are fully redescribed. There are keys to all the genera and to the species within the genera. The descriptions are illustrated by many line drawings in the text by the author and others, and by four fine coloured plates by Mr. Terzi. There is a full account of the characters that are used in classification and of the techniques for mounting and examining specimens. A brief summary of two pages reviews the relation of African Culicines to disease: "perhaps not more than half a dozen are to be regarded as constituting a serious menace on account of their disease-carrying capacities." And there is a chapter on the zoogeography of Ethiopian Culicines with a table summarizing the known distribution of all the species mentioned.

The complexity of systematics becomes apparent whenever a group of animals is sufficiently closely studied; and this is very evident among the mosquitoes. There are species indistinguishable as adults but differing strikingly as larvae or in the egg, species outwardly identical but with very different genitalia, or quite unlike in general pattern but with genitalia that cannot be distinguished, and species similar in appearance but differing in habit. The identification of mosquitoes can therefore never be an easy matter; but the information provided in the three volumes of this series should enable anyone interested in the subject to identify all but a few of the African species with certainty. The present fine work was only just ready for the press when the author died at an age when many more productive years might still have been expected from him. All those who have to do with mosquitoes must deplore the loss of one of our greatest systematists. *V. B. Wigglesworth.*

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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. „ Beriberi.
Bl. „ Blackwater.
B.R. „ Book Review.
Chl. „ Cholera.
Der. „ Tropical Dermatology.
Diet. „ Deficiency Diseases including Epidemic Dropsy.
Dys. „ Dysentery (Bacillary and Unclassed).
Fev. „ Fevers.
Hel. „ Helminthiasis.
Leish. „ Leishmaniasis.
Lep. „ Leprosy.
Lept. signifies Leptospirosis.
Mal. „ Malaria.
Misc. „ Miscellaneous.
Oph. „ Tropical Ophthalmology.
Pel. „ Pellagra.
Pl. „ Plague.
Rab. „ Rabies.
R.B.F. „ Rat-Bite Fever.
Rep. „ Medical and Sanitary Reports.
R.F. „ Relapsing Fever and other Spirochaetoses.
Sp. „ Sprue.
Tryp. „ Trypanosomiasis.
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Y. & S. „ Yaws and Syphilis.

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Collier, W. A., with Esseveld & Mochtar, 99 (R.F.)
Collignon, E., (105) (Mal.)
Conant, N. F., with Figueroa, 86 (Der.)
Congo Belge, 457 (Lep.), 623 (Pl.), 631 (Tryp.)
Conte, D., with Mazza, Basso, G. & Basso, R., 317 (Tryp.)
Cook, A., 304 (Tryp.)
Cook, A. B., 573 (Leish.)
Cook, J. H., with Rogers & Muir, 20 (Lep.)
Coppa, E., with Pansini, Caccuri & Parise, 33 (Mal.)
Cornejo, A., with Mazza, 577 (Leish.)
Corrêa, A., with Pestana & Pessôa, 264 (Leish.)
Cort, W. W., McMullen, D. B., Olivier, L. & Brackett, S., 44, 379 (Hel.)
—— & Otto, G. F., 476 (Hel.)
Costa, H. de M., with Motta, 699 (Lep.)
Cotterell, G. S., 419 (Misc.)
Coulston, F., 543 (Misc.)
—— & Manwell, R. D., 512 (Mal.)
——, with —— & Counts, 651 (Mal.)
Counts, E., with Manwell & Coulston, 651 (Mal.)
Cousin, Grenierboley & Nguyên-van-Minh, 23 (Lep.)
Cova-Garcia, P., (714) (Mal.)
Covell, G., 228, (558) (Mal.), 359 (B.R.)
——, with Christophers & Sinton, 360 (B.R.)
Cowdry, E. V., 23 (Lep.)
Cowen, D., with Wolf & Paige, 354 *bis* (Misc.)
Craig, C. F., 271 (Am.)
Cram, E. B., 524 (Hel.)
Crawford, P. T., with Sanford & Warr, 337 (Mal.)
Cronkite, A. E. & Lack, A. R., 352 (Misc.)
Cruz, J. N., with de Ocampo, 605 (Bb.)
Culbertson, J. T., 314 (Tryp.)
——, with Rose, 111 (Hel.)
Cullyford, J. S., with Topping & Davis, 694 (Fev.)
Culwick, A. T. & Culwick, G. M., 180 (Misc.)
Curbelo, A., Vela, E. M. & Moreno, A., 442 (Fev.)
Cyprus, 355 (Misc.)

D.

Dang-van-Ngu, with Galliard, (733) (Misc.)
——, with —— & Phan-Huy-Quat, 45 (Hel.)
Daniaud, J., with Stefanopoulo, 156 (Hel.)
Daniel, R. A., 177 (Mal.)
Dao van Ty, with Ristorcelli, (696) (Fev.)
Darriba, A. R., 679 (Fev.)
Das Gupta, A. C., with Lal, 606 (Diet.)
——, with ——, Mukherji & Chatterji, 288 (Diet.)
Das Gupta, B. M. & Siddons, L. B., 715 *bis* (Mal.)
Das Gupta, C. C., 652 (Bl.)
Das Gupta, C. R., with Napier, 182 (Misc.)
——, with —— & Rao, 521 (Hel.)
Dassanayake, W. L. P., 152 (Hel.)
Davey, T. F., 21, 706 (Lep.)
Davies, A. & Wingfield, A., 572 (Leish.)
Davis, D. H. S., 622 (Pl.)
Davis, G. B., with Gelfand, (40) (Hel.)
Davis, G. E., 689 (Fev.)
——, with Topping & Cullyford, 694 (Fev.)
Davison, A. R., 466, 705 (Lep.)
Dawson, W. T., with Sanders, (34) (Mal.)
Day, H. W., with Sawyer & Liggett, 527 (Hel.)
De, N. K., 27 (Lep.)
DeEds, F., with McNaught & Beard, 159 (Hel.)
Degotte, J., 458 (Lep.)
De la Barrera, J. M., 324 (Pl.)
Del Campo, G., with d'Ignazio, 54 (Y. & S.)
Del Toro, G., Jr., with Kenrick, 239 (Misc.)

DeMonte, A. J. H., with Chopra & Chatterji, 629 (Pl.)
—— & Gupta, S. K., 480 (Lept.)
Densen, P. M., with Keller & Leathers, 474 (Hel.)
DeOreo, G. A., with Cole, Driver, Johnson & Schwartz, 178 (Mal.)
Derrick, E. H., with Freeman, Brown, Smith & Johnson, 208 (Fev.)
——, Smith, D. J. W. & Brown, H. E., 447 (Fev.)
Desportes, C., 155 (Hel.)
Dévé, F., 471 (Hel.)
Dewan, J. G., 717 (Pel.)
Dharmendra & Bose, R., 702 (Lep.)
—— & Lowe, J., 217 (Lep.)
——, with ——, 462 (Lep.)
Dhondy, B. S., 234 (Misc.)
Dias, E., 639 (Tryp.)
——, with Pifano, 639 (Tryp.)
Días-Rubio, M., 716 (Pel.)
Diaz Atiles, A., 101 (R.F.)
Dick, G. F., with Childs, 393 (Sp.)
Dikshit, B. B. & Ganapathi, K., 647 (Mal.)
——, with Wagle, Sokhey & Ganapathy, 628 (Pl.)
Dill, D. B., with Forbes & Hall, 357 (Misc.)
——, with Robinson, Wilson & Nielsen, 671 (Misc.)
Diwany, M., 347 (Misc.)
Dobell, C., 422 (Misc.)
Dodero, J., 162 (Rab.)
Dods, L., with Walker, 692 (Fev.)
Dollfus, R. P., 112 (Hel.)
Dombrousky, A. I., (517) (Hel.)
Dormer, B. A. & Friedlander, J., 729 (Misc.)
Dowds, J. H., 343 (Bl.), 442 (Fev.)
Dreosti, A. O., (471) (Hel.)
Drevermann, E. B., with Bick, 654 (Bl.)
Driver, J. R., with Cole, DeOreo, Johnson & Schwartz, 178 (Mal.)
Duffalo, J. A., with Hesdorffer, 691 (Fev.)
Dulaney, A. D. & Stratman-Thomas, W. K., 411, 412 (Mal.)
——, with ——, 336, 411 (Mal.)
Durand, P. & Giroud, P., 686 (Fev.)
Duren, A., 306 (Tryp.)
Dussert, E., Faiguenbaum, J. & Neghme, A., 83 (Tryp.)
Dwyer, J. M. & Atkinson, N., 449 (Fev.)
Dy, F. J., with Africa & Soriano, 415 (Mal.)
Dyer, R. E., 681 (Fev.)
Dyer, R. E., Topping, N. H. & Bengtson, I. A., 691 (Fev.)
Dzhangirov, K. G., 179 (Mal.)

E.

Eagle, H., Hogan, R. B., Mohr, C. F. & Black, S. H., 703 (Lep.)
Earle, K. V., 416, 666 *bis* (Misc.)
East African Medical Journal, 432 (Y.F.)
Edwards, F. W., 737 (B.R.)
Edwards, L. B., with Marshall, Bratton & Walker, 597 (Dys.)
Efremov, V. V., 405 (Pel.)
Elles, N. B., 724 (Oph.)
Ellis, A. G., 481 (Misc.)
Elvehjem, C. A., 398 (Pel.)
Enders, J. F., with Liu & Snyder, 681 (Fev.)
Epstein, H. J., with Rothman, 594 (Am.)
Erf, L. A. & Rhoads, C. P., 389 (Sp.)
Ermakova, N. I., 25 (Lep.)
Eskey, C. R., & Haas, V. H., 324 (Pl.)
Esseveld, H., Collier, W. A. & Mochtar, A., 99 (R.F.)
—— & Kouwenaar, W., 449 (Fev.)
——, with ——, 686 (Fev.)
Evans, F. L., 461 (Lep.)

F.

Faiguenbaum, J., with Dussert & Neghme, 83 (Tryp.)
Fairley, N. H., 339 (Bl.)
Fales, J. H., with Sullivan & Goodhue, 235 (Misc.)
Fan, C. & Ch'en, W., 543 (Misc.)
Fan, Y. C., with Hsü, T'an & Ch'in, 118 (Hel.)
Fantus, B., with Traut, E. F. & Greenebaum, R. S., 284 (Diet.)
Farid, M., 710 (Mal.)
Farinaud, M. E., 108 (Mal.)
Farness, O. J., 726 (Oph.)
Faust, E. C., 590 (Am.)
——, with Andrews & Watson, (32) (Mal.)
—— & de Groat, A., 118 (Hel.)
—— & Parker, V., 173 (Mal.)
Fawcitt, J. & Walters, A. H., 508 (Mal.)
Federated Malay States, 35, 102 (Mal.), 153 (Hel.)
Fehily, L., 282 (Bb.)
Feldberg, W., (663) (Vms.)
Feng, L.-C., (32) (Mal.)
Fermin, E. N., with Sison, 284 (Bb.)
Fernández, J. M., with Büngeler, 219 (Lep.)
Fester, A., with Gavrilof, (30) (Lep.)
Fidler, H. K., Glasgow, R. D. & Carmichael, E. B., 166 (Vms.)
Field, J. W., 35, 501 (Mal.)
—— & Le Fleming, H., 501 (Mal.)
—— & Niven, J. C., 107 (Mal.)
Figueroa, H. & Conant, N. F., 86 (Der.)
Findlay, G. M., 431 (Y.F.)
—— & MacCallum, F. O., 68 *bis* (Y.F.)
Finlayson, M. H. & Grobler, G. M., 687 (Fev.)
——, —— & Smithers, R., 205 (Fev.)
Finochietto, R. & Aguilar, H. D., 112 (Hel.)
Fisher, A. A., 659 (Vms.)
Fisher, S., with Franks, 313 (Tryp.)
Fite, G. L., 29 (Lep.)
——, with Badger, 29 (Lep.)
——, with ——, Patrick & Wolfe, 22 (Lep.)
Flecker, H., 166 (Vms.)
Flynn, J. C., 164 (Rab.)
Földes, F., 583 (Chl.)
Foley, E. J., 56 (Y. & S.)
Foley, M. P., Love, J. G., Broders, A. C. & Heilman, F. R., 728 (Misc.)
Foner, A., with Adler, 590 (Am.)
da Fonseca, F., 66 (Y.F.)
—— & Artigas, P., 67 (Y.F.)
Fonte, J., 699 (Lep.)
Forbes, S. B., 177 (Mal.)
Forbes, W. H., Dill, D. B. & Hall, F. G., 357 (Misc.)

Ford, E., 536 (Misc.)
Formijne, P., 668 (Misc.)
Fornaroli, P., with Baserga, 400 (Pel.)
Fossen, A., 170 (Vms.)
Fournier, J., 212 (Chl.)
Fox, J. P. & Gard, S., 69 (Y.F.)
Foy, H. & Kondi, A., 344 (Bl.)
Franck, H., (38) (Hel.)
Franks, A. G. & Fisher, S., 313 (Tryp.)
Frazer, A. C. & Stewart, H. C., 661 (Vms.)
Freeman, B. S., Schrek, R. & Brown, P. F., 591 (Am.)
Freeman, M., with Burnet, 688, 689 (Fev.)
——, Derrick, E. H., Brown, H. E., Smith, D. J. W. & Johnson, D. W., 208 (Fev.)
Freire, R. S., with Mazza, 316 *bis* (Tryp.)
Freund, R. & Samuelson, S., 538 (Misc.)
Friedheim, E. A. H., 634 (Tryp.)
Friedlander, J., with Dormer, 729 (Misc.)
Frimodt-Möller, C., 539 (Misc.)
—— & Barton, R. M., 539 (Misc.)

G.

Gabaldon, A., Antonio Lopez, J. & Ochoa-Palacios, M., 561 (Mal.)
——, Ochoa-Palacios, M. & Perez-Vivas, M. A., 562 (Mal.)
Galliard, H., 117 *bis* (Hel.), 570 (Leish.)
—— & Dang-van-Ngu, (733) (Misc.)
——, —— & Phan-Huy-Quat, 45 (Hel.)
Ganapathi, K., with Dikshit, 647 (Mal.)
——, with Rao, 541 (Misc.)
Ganapathy, K., with Wagle, Sokhey & Dikshit, 628 (Pl.)
Ganguly, S. K., with Roy, 532 (Misc.)
Garcia, E. Y., Navarro, R. J. & Bautista, L., 43 (Hel.)
Gard, S., with Fox, 69 (Y.F.)
Gardner, A., 557 (Mal.)
Garnham, P. C. C., 176 (Mal.)
Gasić Livacić, G., (80), 82 (Tryp.)
—— & Bertìn, V., 82 (Tryp.)
—— & Chait, P., 80 (Tryp.)
Gast Galvis, A., with Smith, Roca García & Calderón Cuervo, 70 (Y.F.)
Gatto, I., 259 (Leish.)
Gaubert, with Giraud, 258 (Leish.)
Gavrilof, W. & Fester, A., (30) (Lep.)
Gay, A. E., with Niño & Pons, 729 (Misc.)
Gayed, I., with Kamal & Anwar, 626 (Pl.)
Gear, J., 450 (Fev.)
Gehr, E., with Oberdoerffer, 22 (Lep.)
Geiger, J. C., 164 (Rab.)
Geiman, Q. M., 264 (Leish.), 695 (Fev.)
Gelfand, M. & Davis, G. B., (40) (Hel.)
George, P. V. & Timothy, B., 621 (Pl.)
Ghosh, B. N. & Kundu, N. L., (170) (Vms.)
Ghosh, H., 272 (Am.)
—— & Chakraborty, R. K., 585 (Chl.)
Ghosh, S. M., with Chopra & Roy, 234, 565, 645 (Mal.)
——, with Roy, 531 (Misc.)
Gibbons, R. J. & Humphreys, F. A., 326 (Pl.)
Giemsa, G. & Nauck, E. G., 119 (Misc.)
Gill, C. A., 410 (Mal.)
Gillette, D. F., 724 (Oph.)
Gilmore, R. M., with Bugher, Boshell-Manrique & Roca-Garcia, 434 (Y.F.)
Gingrich, W. D., 650 *bis* (Mal.)
Giraud, P. & Gaubert, 258 (Leish.)
Giri, K.V., with Naganna & Venkatesam, 721 (Pel.)
Giroud, P., with Durand, 686 (Fev.)
Glasgow, R. D., with Fidler & Carmichael, 166 (Vms.)
Gminder, E., 27 (Lep.)
Goenawan, R., with Soetopo, 52 (Y. & S.)
Gold Coast, 636 (Tryp.)
Gomez, F., with Navarro, 54 (Y. & S.)
González, L. M., with Krakower, 28 (Lep.)
Goodale, R. H., with Welcker & Kaneb, 472 (Hel.)
Goodhue, L. D., with Sullivan & Fales, 235 (Misc.)
Goyal, R. K., 680 (Fev.)
Graham, C. F., 525 (Hel.)
Graham, R., Morrill, C. C. & Boley, L. E., 160 (Rab.)
Granett, P., 713 (Mal.)
Grant, H. M., with Spies & Grant, J. M., 404 (Pel.)
Grant, J. M., with Spies & Grant, H. M., 404 (Pel.)
Grasset, E. & Schaafsma, A. W., 660 (Vms.)
Gray, H. F., with Herms, 58 (B.R.)
Gray, J. K., with McMullen, 588 (Am.)
Green, R. A., Breazeale, E. L. & Harding, H. B., 203 (Fev.)
Greene, E. I. & Greene, J. M., 525 (Hel.)
Greene, R. A., with Andes & Breazeale, 158 (Hel.)
Greenebaum, R. S., with Fantus & Traut, 284 (Diet.)
Greengrass, J. J. & Silverman, I., (466) (Lep.)
Greig, E. D. W., 40 (Hel.)
Grenierboley, with Cousin & Nguyên-van-Minh, 23 (Lep.)
Greval, S. D. S. & Nicholas, M. J., 165, 497 (Rab.)
Griasnow, N. & Suslowa, M., 495 (Rab.)
de Groat, A., with Faust, 118 (Hel.)
Grobler, G. M., with Finlayson, 687 (Fev.)
Grobler, J. M., with Finlayson & Smithers, 205 (Fev.)
Gromaschewski, L. & Morozkin, N., 441 (Fev.)
Grundmann, A. W., with Ackert & Boles, 623 (Pl.)
Guenette, J. A., with Miller, Choquette, Audet & Kelso, 526 (Hel.)
Guerra, P., 201 (Fev.), 353 (Misc.)
Guidoni, P., with Radaody-Ralarosy, 151 (Hel.)
Guillier, G. & Radaody-Ralarosy, P., 84 (Der.)
Guindy, 212 (Chl.), 410 (Mal.), 454 (Fev.)
Gunewardene, S. R., 38 (Hel.)
Gunther, C. E. M., 445 (Fev.)
Gupta, S. K., with deMonte, 480 (Lept.)
Gutierrez, M., 604 (Bb.)

H.

Haas, V. H., with Eskey, 324 (Pl.)
Habel, K., 161, 162, 495 (Rab.)
Hackett, L. W., 102 (Mal.)

Haden, R. L., 342 (Bl.)
Hahn, P. F., Ross, J. F., Bale, W. F. & Whipple, G. H., 116 (Hel.)
Haight, C. & Alexander, J., 111 (Hel.)
Halawani, A., 263 (Leish.)
Halder, K. C., with Smith & Ahmed, 256 (Leish.)
Hall, F. G., with Forbes & Dill, 357 (Misc.)
Hallauer, C. & Kuhn, H., 97 (R.F.)
Ham, C., with Young, 375 (Hel.)
Hamilton, D. G., 186 (Misc.)
Hamilton, J. B., 346 (Oph.)
Hand, R. F., 16 (Sp.)
Hanes, F. M. & Reiser, R., 389 (Sp.)
Hansman, F. S., 18 (Sp.)
Haralson, M. F., 441 (Fev.), 626 (Pl.)
Harding, H. B., with Green & Breazeale, 203 (Fev.)
Harding, R. D., 75, 76 (Tryp.)
Hardwick, C., with Bennett, 7 (Sp.)
Hare, K. P., 182, 183 (Misc.)
Harris, B. P., 522 (Hel.)
Harris, S., (401) (Pel.)
Hart, L., 607 (Diet.)
Hartree, E. F., with Keilin, 656 (Bl.)
Harvey, W. F., 416 (Misc.)
Hasseltine, H. E., 458 (Lep.)
Hassin, G. B., 683 (Fev.)
Hawaii, 441 (Fev.), 626 (Pl.)
Hawking, F., 78, 306, 312 *bis*, 633 (Tryp.), 146, 147, 151 *bis*, 522 (Hel.), 352 (Misc.)
Hayashi, F., with Igarashi, 463 (Lep.)
Haynes, W. S., 227 (Mal.)
Hayter, R. T. M., with Chopra & Sen, 34 (Mal.)
——, with ——, —— & Talukdar, 34, (713) (Mal.)
Hayward, E. W., 456 (Lep.)
Heaslip, W. G., 445, 451 (Fev.)
Hegner, R., 296 (Mal.)
——, Beltrán, E. & Hewitt, R., 589 *bis* (Am.)
Heilman, F. R., with Foley, Love & Broders, 728 (Misc.)
Heisch, R. B., 230 (Mal.)
Hemingway, M. W., Hemingway, R. W. & Arneson, V. K., 479 (R.F.)
Hemingway, R. W., with Hemingway, M. W. & Arneson, 479 (R.F.)
Henderson, H. J., 222 (Lep.)
Henderson, J. M. & Howard, R. S., Jr., 292 (Mal.)
Henderson, R. G., with Pinkerton, 533 (Misc.)
Hennessey, R. S. F., with Burton, 329 (Pl.)
Henriksen, S. D. & Oeding, P., 734 (B.R.)
Hermans, E. H., 52 (Y. & S.)
Herms, W. B. & Gray, H. F., 58 (B.R.)
Hernandez Morales, F., with Rodríguez Olleros, 15 (Sp.)
Heron, G. W., 164, 497 (Rab.)
Herron, P. H., 497 (Rab.)
Hertig, M., 236 (Misc.)
Hesdorffer, M. B. & Duffalo, J. A., 691 (Fev.)
Hester, K. H. C., with Martin, 537 (Misc.)
Hewitt, R., 120 (B.R.), 295 (Mal.)
——, with Hegner & Beltran, 589 *bis* (Am.)
——, with Otto & Strahan, 473 (Hel.)
Hicks, C. S., 672 (Misc.)
Hill, R. B. & Cambournac, F. J. C., 564 (Mal.)
Ho, E. A., Chu, H. J. & Yuan, I. C., 571 (Leish.)
Hoare, C. A., 314 (Tryp.)
Hobmaier, A. & Hobmaier, M., 239 (Misc.)
Hodes, H. L., Webster, L. T. & Lavin, G. I., 161 (Rab.)
Hodge, E. H. V., (601) (Dys.)
Hodgkin, E. P., 153 (Hel.)
Hoeppli, R., 252 (Leish.)
—— & Ch'iang, I., (39) (Hel.)
Hoff, H. & Shaby, J. A., 56 (Y. & S.)
Hoffmann, E., (54) (Y. & S.)
Hoffmann, W. H., 222 (Lep.)
Hogan, R. B., with Eagle, Mohr & Black, 703 (Lep.)
Hollaender, A., Jones, M. F. & Jacobs, L., 526 (Hel.)
——, with —— & ——, 527 (Hel.)
Hollands, R. A., with Mateer, Baltz & Marion, 270 (Am.)
Holt, L. E., Jr., with Najjar, 400 (Pel.)
Hood, M., 594 (Am.)
van Hoof, L., 457 (Lep.), 623 (Pl.), 631 (Tryp.)
Hopkins, G. H. E., 233 (Mal.), 629 (Pl.)
—— & Chorley, T. W., 98 (R.F.)
Hopkins, J., with Jamison, 534 (Misc.)
Hornibrook, J. W. & Nelson, K. R., 691 (Fev.)
Hou, H. C., 406, 719 (Pel.)
Howard, R. S., Jr., with Henderson, 292 (Mal.)
Howell, D. E. & Stiles, G. W., 733 (Misc.)
Howell, K. M. & Knoll, E. W., 267 (Am.)
Howitt, B., 494 (Rab.)
Hsü, C., with Chu, 380 (Hel.)
Hsü, H. F., 384 (Hel.)
——, Fan, Y. C., T'an, C. C. & Ch'in, K. Y., 118 (Hel.)
—— & Li, S. Y., 380 *bis* (Hel.)
Hsü, Y. K., 112 (Hel.)
Hu, C. H., 254, 256 (Leish.)
——, with Pai, 571 (Leish.)
Hu, S. M. K., 105, 176 (Mal.), 148, 149 *bis*, 520 (Hel.)
Hua, T. J. & Cheng, S. Y., 652 (Bl.)
Huang, Y. W., Shen, T. H. & Tang, F. F., 579 (Chl.)
Hudson, N. P., 448 (Fev.)
Huff, C. G., 413 (Mal.)
——, with Kitchen & Roe, (36) (Mal.)
——, with Porter, 513 (Mal.)
Huff, N. E., with Spies, Bean & Vilter, 720 (Pel.)
Hughes, L., 18 (Sp.)
Hughes, W., (17) (Sp.)
Hummel, H. G., 272 (Am.)
Humphreys, F. A., with Gibbons, 326 (Pl.)
Hutton, E. L., 333 (Mal.)

I.

Idnani, J. A., with Ray, 453 (Fev.)
Igarashi, M. & Hayashi, F., 463 (Lep.)
Ignacio Chala, J., (221) (Lep.)
d'Ignazio, C. & Del Campo, G., 54 (Y. & S.)
——, Lombardi, A. & d'Arcangelo, D., 200 (Fev.)
Imbert, M., with Sabadini, 39 (Hel.)

India, 228 (Mal.)
International Journal of Leprosy, 27, 222 (Lep.)
Ishii, N., 254 (Leish.)
——, Sawada, T. & Shimizu, S., 254, 258 (Leish.)
Itakura, T., 218 *bis*, (700) (Lep.)
Iwata, S., (473) (Hel.)
Iyengar, M. O. T., 228 (Mal.)
Iyer, P. V. S., with Shortt & Menon, 413 (Mal.)

J.

Jack, R. W., 307 (Tryp.)
Jackson, C. H. N., 74, 307, 632 (Tryp.)
Jacobi, L., 566 (Mal.)
Jacobs, L., with Hollaender & Jones, 526 (Hel.)
——, with Jones & Hollaender, 527 (Hel.)
James, C., 664 (Misc.)
Jamison, S. C. & Hopkins, J., 534 (Misc.)
Janer, J. L., 378 (Hel.)
Jean, J. T., with Tsuchiya, 267 (Am.)
Jersild, M., 398 (Pel.)
Jiménez Garcia, F., 401 (Pel.)
Jiminez, J. F. & Buddingh, G. J., 453 (Fev.)
Jírovec, O. & Rodová, H., 733 (Misc.)
Jobbins, D. M., with Boyd, 106 (Mal.)
——, with Clark & Komp, 511 (Mal.)
John, C. C., 233 (Mal.)
Johnson, C. M., 591 (Am.)
Johnson, D. W., with Freeman, Derrick, Brown & Smith, 208 (Fev.)
Johnson, H. H., with Cole, DeOreo, Driver & Schwartz, 178 (Mal.)
Johnson, H. N. & Leach, C. N., 159, 496 (Rab.)
——, with Leach, 164, 496 (Rab.)
Jolliffe, N., (286) (Bb.)
——, with Most, 175 (Mal.)
Jolly, G. G., 725 (Misc.)
Jones, M. F., with Hollaender & Jacobs, 526 (Hel.)
——, Jacobs, L. & Hollaender, A., 527 (Hel.)
Jörg, M. E., with Mazza, 318 (Tryp.)
Journal of the American Medical Association, 284 (Bb.), 350 (Misc.), 526 (Hel.)
Journal of the Egyptian Public Health Association, 629 (Pl.)
Joyeux, C. & Baer, J. G., 113 (Hel.)
Juler, F. A., with Brooks & Williams, 346 (Oph.)
Junior, F. P., 323 (Pl.)
—— & Arantes, J. B., 662 (Vms.)
Junior, M. S. & de Albuquerque, R., 628 (Pl.)

K.

Kaku, S., 203 (Fev.)
Kamal, A. M., 626 (Pl.)
——, Gayed, I. & Anwar, M., 626 (Pl.)
Kaneb, G. D., with Welcker & Goodale, 472 (Hel.)
Kariadi, 520 (Hel.)
Karunakaran, C. O. & Nair, P. K., 286 (Bb.)
Kasahara, M. & Sha-Shi-Nan, 496 (Rab.)
Kawahigasi, K., 685 (Fev.)
Keilin, D. & Hartree, E. F., 656 (Bl.)
Kekhcher, O. M., 503 (Mal.)
Kellaway, C. H. & Trethewie, E. R., 168 (Vms.)
Keller, A. E., Leathers, W. S. & Densen, P. M., 474 (Hel.)
Kelsey, A. J., 418 (Misc.)
Kelso, R. F., with Miller, Choquette, Audet & Guenette, 526 (Hel.)
Kenrick, G. W. & Del Toro, G., Jr., 239 (Misc.)
Kessel, J. F., 727 (Oph.)
Ketchum, W. H., with Campbell, 206 (Fev.)
Kikuth, W., 636 (Tryp.)
Kingsbury, A. N., 102 (Mal.)
Kinugasa, M., 45 (Hel.)
Kirk, R. & Macdonald, D. R., 261 (Leish.)
—— & Sati, M. H., 260, 261, 573 (Leish.)
Kitchen, S. F., Huff, C. G. & Roe, M. A., (36) (Mal.)
Kligler, I. J. & Bernkopf, H., 494 (Rab.)
——, with Mer & Birnbaum, 562 (Mal.)
Klüver, C., with Taliaferro, 412 *bis* (Mal.)
Knipe, F. W., with Russell, 645 (Mal.)
——, with —— & Rao, 564 (Mal.)
Knoll, E. W., with Howell, 267 (Am.)
Knott, F. A. & Leibel, B., 665 (Misc.)
Kobayashi, H., 520 (Hel.)
Kobayasi, H., 149 (Hel.)
Kobro, M., with Salvesen, 17 (Sp.)
Kochhar, B. D., (401) (Pel.)
Kolodny, M. H., 81 (Tryp.)
Komiya, Y. & Tajimi, T., 44 (Hel.)
Komp, W. H. W., 106 (Mal.)
——, with Clark & Jobbins, 511 (Mal.)
——, with Kumm & Ruiz, (352) (Misc.)
Kondi, A., with Foy, 344 (Bl.)
Kooser, J. H. & Blankenhorn, M. A., 717 (Pel.)
Kourí, P., with Rappaport, I., 113 (Hel.)
Kouwenaar, W. & Esseveld, H., 686 (Fev.)
——, with Esseveld, 449 (Fev.)
Krakower, C. & González, L. M., 28 (Lep.)
Krakower, C. A., 376 (Hel.)
Krueger, A. P. & Scribner, E. J., 586 (Chl.)
Kudicke, R., 225 (Lep.)
Kuhn, H., with Hallauer, 97 (R.F.)
Kuitunen-Ekbaum, E., 156, 524 (Hel.), 589 (Am.)
Kumm, H. W., Komp, W. H. W. & Ruiz, H., (352) (Misc.)
——, with Peña Chavarria, 351 (Misc.)
Kundu, N. L., with Ghosh, (170) (Vms.)
Kurotchkin, T. J. & Wyckoff, R. W. G., 451 (Fev.)
Kuznezov, V. N., 705 (Lep.)
Kwee tat Tjhong, 481 (Lept.)

L.

L'Abbate, G., 40 (Hel.)
La Barge, O. J., 592 (Am.)
Lacaz, C. da S., 93 (Der.)
Lack, A. R., with Cronkite, 352 (Misc.)
Laha, P. N., 259 (Leish.)
Lahiri, B. N., with Webster, McGuire, Stephens & Lahiri, 164 (Rab.)

Lal, R. B., Chatterji, S. R. & Agarwala, S. P., 607 (Diet.)
—— & Das Gupta, A. C., 606 (Diet.)
——, Mukherji, S. P., Das Gupta, A. C. & Chatterji, S. R., 288 (Diet.)
Lal, S., with Pasricha, 13 (Sp.)
——, with —— & Malik, 608 (Diet.)
Lamborn, W. A., 22 (Lep.), 72 (Tryp.)
Lampe, P. H. J., 94 (Der.)
Lamprell, B. A., 179 (Mal.)
Lancet, (432) (Y.F.), 440, 687 (Fev.)
Landor, J. V., 95 (Der.)
Lane, C., 114 (Hel.)
de Langen, C. D., 388 (Sp.)
Larenas, R., with Beltran, 567 (Mal.)
Larios Rodriguez, I., 383 (Hel.)
Lavin, G. I., with Hodes & Webster, 161 (Rab.)
Laws, F., with Swan, 285 (Bb.)
Lazuk, A. D., 510 (Mal.)
Leach, C. N. & Johnson, H. N., 164, 496 (Rab.)
——, with Johnson, 159, 496 (Rab.)
League of Nations, 578 (Chl.), 619 (Pl.), 679 (Fev.)
Leathers, W. S., with Keller & Densen, 474 (Hel.)
Lederer, R., 417 (Misc.)
Ledger, L. K. & Wasson, T. C., 725 (Misc.)
Lee, D. H. K., 544 (Misc.)
—— & Boissard, G. P. B., 546 (Miscc.)
Le Fleming, H., with Field, 501 (Mal.)
Le Gac, P. & Lepesme, P., 167 (Vms.)
Lehucher, P., with Anderson, 155 (Hel.)
Leibel, B., with Knott, 665 (Misc.)
Leimena, J., 685 (Fev.)
Lennox, F. G., 420 (Misc.)
—— & Webber, L. G., 420 (Misc.)
de Leon, A. D., 712 (Mal.)
León y Blanco, F., 88, 89 *bis*, 90 *bis*, (91), 91 *ter* (Der.)
Leong, P. C., 285 (Bb.)
Lepesme, P., with Le Gac, 167 (Vms.)
Leprosy in India, 698 (Lep.)
Le Roy & Chertin, 16 (Sp.)
LeVan, J. H., 70 *bis* (Y.F.)
Levin, J., 449 (Fev.)
Levy, A. H., 155 (Hel.)
Li, S. Y., with Hsü, 380 *bis* (Hel.)
Li, T. C., with Chung, 570 (Leish.)
Li, Y., 516 (Hel.)
Lie Kian Joe, 159 (Hel.)
——, with Bonne, 469, 475 (Hel.)
Liem Tjaij Tie, 711 (Mal.)
Lieu, V. T., 255 (Leish.)
Liggett, R. S., with Sawyer & Day, 527 (Hel.)
Lillie, R. D., Perrin, T. L. & Armstrong, C., 446 (Fev.)
Lin, C. C., with Chang, 375 (Hel.)
Linton, R. & Sarkar, N., 662 (Vms.)
Linton, R. W., 580 (Chl.)
Lippelt, H., with Mohr, 518 (Hel.)
Litchfield, J. T., Jr., with Marshall, Bratton & White, 596 (Dys.)
Liu, H-L., 593 (Am.)
Liu, P. Y., with Pang & Zia, 207 (Fev.)
——, Snyder, J. C. & Enders, J. F., 681 (Fev.)
—— & Zia, S. H., 207 (Fev.)
Liu, W.-T., Chung, H.-L. & Zia, S. H., 202 (Fev.)
Lleras Restrepo, with Chala & Brumpt, 24 (Lep.)
Lob, S., with Sawitz, d'Antoni & Rhude, 523 (Hel.)
Loh, G. L., with MacGregor, 669 (Misc.)
——, with —— & Richards, 349 (Misc.)
Lombardi, A., with d'Ignazio & d'Arcangelo, 200 (Fev.)
Loos, D., 480 (Lept.)
Love, J. & Tayloe, G. B., 276 (Dys.)
Love, J. G., with Foley, Brider & Heilman, 728 (Misc.)
Loving, W. L., 700 *bis* (Lep.)
Lowe, J. & Chatterji, S. N., 222, 700, 701 *ter* (Lep.)
—— & Dharmendra, 462 (Lep.)
——, with ——, 217 (Lep.)
—— & Santra, I., 21 (Lep.)
Ludlam, G. B., Batchelor, I. R. C. & Rhodes, A. J., 683 (Fev.)
Lumsden, W. H. R. & Bertram, D. S., 414 *bis* (Mal.)
Lyle, T. K. & McLean, D. W., 723 (Oph.)
Lynch, P. P. & Brandt, C. W., 291 (Mal.)
Lyon, G. M., 599 (Dys.)
Lyster, L. L., 158 (Hel.)

M.

MacCallum, F. O., with Findlay, 68 *bis* (Y.F.)
McCarthy, D. D., 547 (B.R.)
McCay, F. & White, R. S., 531 (Misc.)
Macchiavello, A., 624 (Pl.)
McCoy, O. R., 529 (Hel.)
Macdonald, D. R., with Kirk, 261 (Leish.)
McGrath, J., O'Farrell, P. T. & Boland, S. J., 274 (Dys.)
MacGregor, R. G. S. & Loh, G. L., 669 (Misc.)
——, Richards, W. & Loh, G. L., 349 (Misc.)
McGuire, J. P., with Webster, Stephens & Lahiri, 164 (Rab.)
McKean, J. H., with Collier, 26 (Lep.)
McKenzie, A., 722 (Pel.)
Mackie, T. T. & Mills, M. A., 390 (Sp.)
McLean, D. W., with Lyle, 723 (Oph.)
McLetchie, J. L., 76 (Tryp.)
McMahon, J. P., 154 (Hel.)
McMullen, D. B., with Cort, Olivier & Brackett, 44, 379 (Hel.)
—— & Gray, J. K., 588 (Am.)
McMurdo, H. B., 333 (Mal.)
McNally, A. & Case, J. B., (472) (Hel.)
McNaught, J. B., Beard, R. R. & DeEds, F., 159 (Hel.)
Macnie, J. P., 724 (Oph.)
Madras, 347 (Oph.)
de Magalhães, O., 444 *bis* (Fev.)
—— & Moreira, J. A., 443 (Fev.)
—— & Penna, R., 444 (Fev.)
Mahaffy, A. F., with Smithburn & Paul, 416 (Misc.)
Mail, G. A., 205 (Fev.)
Majid, A., with Afridi & Shah, 105 (Mal.)
Makand, S. N., 579 (Chl.), 621 (Pl.)
Malaihollo, J. F., 457 (Lep.)
Malik, K. S. & Pasricha, C. L., 582 (Chl.)
——, with —— & Lal, 608 (Diet.)
Malkiel, B. P., 643 (Mal.)

Maltaner, E., 463 (Lep.)
Manalang, J., 698 (Lep.)
Manchanda, S. S., with Taylor, 184 (Misc.)
Manson-Bahr, P., 7, 386 (Sp.), 333 (Mal.), 516 (Hel.), 415, 529 (Misc.)
—— & Walton, J., 382 (Hel.)
Manwell, R. D., 513 (Mal.)
——, with Coulston, 512 (Mal.)
——, Counts, E. & Coulston, F., 651 (Mal.)
Maplestone, P. A. & Bhaduri, N. V., 374 (Hel.)
—— & Mukerji, A. K., 114 (Hel.)
——, with Panja, 95 (Der.)
——, with Rao, 152 (Hel.)
Mapother, P., with Moore, (93) (Der.)
Mara, L., with Brambilla & Campisi, 708 (Mal.)
Marberg, K., with Reitler, 280 (Dys.)
Marchionini, A., 263 (Leish.)
Mariani, G., 199 (Fev.)
Marion, D. F., with Mateer, Baltz & Hollands, 270 (Am.)
Markoff, N., 391 (Sp.)
Marshall, E. K., Jr., Bratton, C., Edwards, L. B. & Walker, E., 597 (Dys.)
——, ——, White, H. J. & Litchfield, J. T., Jr., 596 (Dys.)
Martin, J. T. & Hester, K. H. C., 537 (Misc.)
Martins, A. F., 259 (Leish.)
Martins, A. V., 42 (Hel.), 639 (Tryp.)
—— & Tupynamba, A., 639 (Tryp.)
Maru, A. M., 542 (Misc.)
Mason, E. D., 423 (Misc.)
Mason, J. H. & Alexander, R. A., 203 *bis* (Fev.)
——, with Alexander, 203 (Fev.)
——, with —— & Neitz, 203 (Fev.)
Masunaga, E., with Badger & Wolf, 29 (Lep.)
Mateer, J. G., Baltz, J. I., Marion, D. F. & Hollands, R. A., 270 (Am.)
Mathews, G. B., with Tchang, 450 (Fev.)
Matuda, S., 517 (Hel.)
Mauss, E. A., 528 *bis* (Hel.)
Mauzé, J., with Berny, 24 (Lep.)
May, A. J., 685 (Fev.)
Mayer, M. & Pifano, F., 640 (Tryp.)
Mazza, S., 251 (Leish.), 315 (Tryp.)
——, Basso, G. & Basso, R., 318 (Tryp.)
——, ——, —— & Conte, D., 317 (Tryp.)
—— & Cornejo, A., 577 (Leish.)
—— & Freire, R. S., 316 *bis* (Tryp.)
—— & Jörg, M. E., 318 (Tryp.)
—— & Miyara, S., 317, (641) (Tryp.)
——, ——, Basso, G., & Basso, R., (640) (Tryp.)
—— & Urcelay, G., 316 (Tryp.)
Mazza, S., & others, 319, (640), (641) (Tryp.)
Mazumdar, D. C., with Roy & Mukherjee, 421 (Misc.)
Mazzotti, L., 81 (Tryp.), 731 (Misc.)
——, with Osorio, 525 (Hel.)
de Meillon, B., 185 (Misc.)
Meinardi-Flaumenhaft, T., 537 (Misc.)
Meira, J. A., 378 (Hel.)
—— & Amaral, A. D. F., 375 (Hel.)
Meleney, H. E., 355 (Misc.), 681 (Fev.)
——, with Snyder, 589 (Am.)
Mello, G. B., 571 (Leish.)
Menon, K. P., with Shortt, 338 *bis* (Mal.)
——, with —— & Iyer, 413 (Mal.)
Menon, M. A. U., (153) (Hel.)
Menon, T. B. & Ramamurti, B., 519 (Hel.)
Mer, G., Birnbaum, D. & Kligler, I. J., 562 (Mal.)
Mertens, W. K. & Beeuwkes, H., 215 (Chl.)
Mettier, S. R., with Buell, 655 (Bl.)
Meulengracht, E., with Bichel, 406 (Pel.)
Meyer, K. F., (630) (Pl.)
Meyers, F. M., 602 (Bb.)
Middleton, W. M. & Milroy, G., 721 (Pel.)
Miller, M. J. & Choquette, L., 525 (Hel.)
——, ——, Audet, W., Kelso, R. F. & Guenette, J. A., 526 (Hel.)
Mills, M. A., with Mackie, 390 (Sp.)
Milner, J. G., 347 (Oph.)
Milroy, G., with Middleton, 721 (Pel.)
Mitra, S. L., 215 (Chl.), 251 (Leish.)
Miyara, S., with Mazza, 317, (641) (Tryp.)
——, with ——, Basso, G. & Basso, R., (640) (Tryp.)
Mochtar, A., with Esseveld & Collier, 99 (R.F.)
Mohamed, A. S., 40 *bis* (Hel.)
Mohan, B. N., with Mulligan & Russell, 646 (Mal.)
Mohr, C. F., with Eagle, Hogan & Black, 703 (Lep.)
Mohr, W. & Lippelt, H., 518 (Hel.)
Moise, R., 709 (Mal.)
Moiser, B., 704 (Lep.)
Moll, A. A. & O'Leary, S. B., 321, 625 (Pl.)
Monacelli, M. & Pisani, D., 57 (Y. & S.)
Mondal, R. S., 564 (Mal.)
Monnier, A., 441 (Fev.)
——, with Castaneda & Silva, 448 (Fev.)
Mooij, W., 512 (Mal.)
Moore, M., 535 (Misc.)
—— & Mapother, P., (93) (Der.)
Moreau, P., 38 (Hel.)
Moreira, J. A., with de Magalhães, 443 (Fev.)
Moreno, A., with Cúrbelo & Vela, 442 (Fev.)
Morgan, E., 180 (Mal.)
Morozkin, N. I., 440 (Fev.)
——, with Gromaschewski, 441 (Fev.)
Morrill, C. C., with Graham & Boley, 160 (Rab.)
Morrison, S. K. & Parsons, L., 479 (R.F.)
Moseley, V., with Callaway, 83 (Der.)
Most, H., 174 (Mal.)
—— & Jolliffe, N., 175 (Mal.)
—— & Tewkesbury, M. H., 644 (Mal.)
Motta, J. & Costa, H. de M., 699 (Lep.)
Mottoulle, L., (423), 543 (Misc.)
Mreyen, F. W., with Bonne, 112 (Hel.)
Mueller, J. F., 469 (Hel.)
Muir, E., 223 *bis*, 706 (Lep.)
——, with Rogers & Cook, 20 (Lep.)
Mukerji, A. K., with Maplestone, 114 (Hel.)
Mukherjee, B. N., with Tribedi, 92 (Der.)
Mukherjee, P., with Roy & Mazumdar, 421 (Misc.)
Mukherji, S. P., with Lal, Das Gupta & Chatterji, 288 (Diet.)
Mulligan, H. W. & Russell, P. F., 338 (Mal.)
——, —— & Mohan, B. N., 646 (Mal.)
——, Sommerville, T. & Swaminath, C. S., 647, 648 *bis* (Mal.)
—— & Swaminath, C. S., 650 (Mal.)
Munk, F., (201) (Fev.)
Murdock, J. R., 627 (Pl.)

N.

Naganna, B., Giri, K. V. & Venkatesam, P., 721 (Pel.)
Naidu, S. R., 273 (Dys.)
Nair, P. K., with Karunakaran, 286 (Bb.)
Najjar, V. A. & Holt, L. E., Jr., 400 (Pel.)
Nakamura, T., (169) (Vms.)
Nakasato, T., with Yosino, 39 (Hel.)
Nandi, B. K., 645 (Mal.)
Napier, L. E., (17) (Sp.), 183 (Misc.), 333 (Mal.), 437 (Y.F.)
—— & Das Gupta, C. R., 182 (Misc.)
——, —— & Rao, S. S., 521 (Hel.)
—— & Sen, G. N., 574 (Leish.)
——, with Sen Gupta, 289 (Diet.)
Narayan, J., 712 (Mal.)
Narayana, P. A., with White, 561 (Mal.)
Nargund, K. S., with Vyas & Bokil, 376 (Hel.)
Nature, 272 (Am.)
Nauck, E. G., with Giemsa, 119 (Misc.)
—— & Zumpt, F., 443 (Fev.)
Navarro, R. J., 348 (Misc.)
—— & Gomez, F., 54 (Y. & S.)
——, with Garcia & Bautista, 43 (Hel.)
de Navasquez, S., 340 (Bl.)
Neghme, A., with Dussert & Faiguenbaum, 83 (Tryp.)
Negroni, P., 534 (Misc.)
—— & Tey, J. A., 92 (Der.)
Neilson, W. P., 96 (R.F.)
Neitz, W. O., with Alexander & Mason, 203 (Fev.)
Nelson, E. C., 276 (Dys.)
Nelson, K. R., with Hornibrook, 691 (Fev.)
Nguyên-van-Minh, with Cousin & Grenierboley, 23 (Lep.)
Nicholas, M. J., with Greval, 165, 497 (Rab.)
Nicholls, L., 206 (Fev.)
Nicolson, E. L., (517) (Hel.)
Nielsen, M., with Robinson, Dill & Wilson, 671 (Misc.)
Nieto Roaro, D., with Varela, 88 (Der.)
Nigeria, 631 (Tryp.)
Niño, F. L., Pons, L. M. & Gay, A. E., 729 (Misc.)
—— & Rivas, C. I., 237 (Misc.)
Nitto, S., with Ota, 461, 462 (Lep.)
Nitzescu, I. I. & Stan-Suciu, M., 661 (Vms.)
Niven, J. C., with Field, 107 (Mal.)
Nolasco, J. O., 25, 220 (Lep.)
Nonaka, N., 224 (Lep.)
Nucciotti, L., 107 (Mal.)
Nutter, P. B., Rodaniche, E. C. & Palmer, W. L., 595 (Am.)

O.

Oag, R. K., 80 (Tryp.), 97 (R.F.)
Oberdörffer, M., 26 (Lep.)
—— & Gehr, E., 22 (Lep.)
Oberdoerffer, M. J. & Collier, D. R., 220 (Lep.)
de Ocampo, G. & Cruz, J. N., 605 (Bb.)
Ochoa, R. R., with Silva, 682 (Fev.)
Ochoa-Palacios, M., with Gabaldon & Antonio Lopez, 561 (Mal.)
——, with —— & Perez-Vivas, 562 (Mal.)
O'Connor, K. J., 184 (Misc.)
Oeding, P., with Henriksen, 734 (B.R.)
O'Farrell, P. T., McGrath & Boland, 274 (Dys.)
Ogurtsova, A. S., 642 (Mal.)
Ohama, S., 474 (Hel.)
Oláh, E., 346 (Oph.)
O'Leary, S. B., with Moll, 321, 625 (Pl.)
Olitsky, P. K., (454) (Fev.)
Oliver-González, J., 528 (Hel.)
Olivier, L., with Cort, McMullen & Brackett, 44, 379 (Hel.)
Oomen, H. A. P. C., 52 (Y. & S.)
Ormsby, O. S. (401) (Pel.)
Orquiza, C. T., with Chikiamco, 517 (Hel.)
Orsini, O., 576 (Leish.)
Osorio, M. T. & Mazzotti, L., 525 (Hel.)
O'Sullivan, J. F., 394 (Sp.)
Ota, M. & Nitto, S., 461, 462 (Lep.)
Otten, L., 330 (Pl.)
Otto, G. F., with Brown, 476 (Hel.)
——, with Cort, 476 (Hel.)
——, Hewitt, R. & Strahan, D. E., 473 (Hel.)
Otto, H., 278 (Dys.)
Otto R. & Wohlrab, R., 207 (Fev.)
Ou, T., 507 (Mal.)
Ouyang, G., 277 (Dys.)
Overbeek, J. G., (561) Mal.)

P.

Packchanian, A., 81 *bis*, 637 (Tryp.)
Pahati, A. P., 104 (Mal.)
Pai, H. C. & Hu, C. H., 571 (Leish.)
Paige, B. H., with Wolf & Cowen, 354 *bis* (Misc.)
Pakenham-Walsh, R. & Rennie, A. T., 231 (Mal.)
Palawandow, G., 495 (Rab.)
Palestine, 164, 497 (Rab.)
Palit, C. C., Basu, S. N. & Varma, V. L., 608 (Diet.)
Pallister, R. A., (287) (Bb.)
Palmer, P. F., 494 (Rab.)
Palmer, W. L., with Nutter & Rodaniche, 595 (Am.)
Pampana, E. & Casini, G., 710 (Mal.)
Pandit, C. G., with Anderson, Sanjiva Rao & Shortt, 454 (Fev.)
Pang, K. H., Liu, P. Y. & Zia, S. H., 207 (Fev.)
Panja, D. & Maplestone, P. A., 95 (Der.)
Panja, G., with Pasricha, 620 (Pl.)
——, with —— & Paul, 580 (Chl.)
Pannekoek-Westenburg, S. J. E. & van Veen, A. G., 602 (Bb.)
Panneton, P., 723 (Oph.)
Pansini, G., Caccuri, S., Coppa E. & Parise, N., 33 (Mal.)
Paraense, L. & Chagas, A. W., 572 (Leish.)
Pardal, E., 625 (Pl.)
Pardo-Castello, V. 49 (Y. & S.), 89 (Der.)
Parise, N., with Pansini, Caccuri & Coppa, 33 (Mal.)
Parker, R. R., 688 (Fev.)
Parker, V., with Faust, 173 (Mal.)
Parsonnet, A. E. & Bernstein, A., 661 (Vms.)
Parsons, L., with Morrison, 479 (R.F.)

Pasricha, C. L. & Lal, S., 13 (Sp.)
—— & Malik, K. S., 583 (Chl.)
——, with Malik, 582 (Chl.)
——, —— & Lal, S., 608 (Diet.)
—— & Panja, G., 620 (Pl.)
——, —— & Paul, B. M., 580 (Chl.)
Passmore, R. & Sommerville, T., 646 (Mal.)
Patiño Camargo, L., 209, 695 (Fev.), 351 (Misc.)
Patrick, D. W., with Badger, Fite & Wolfe, 22 (Lep.)
Patterson, T. C., 531 (Misc.)
Patwardhan, N. G., with Pradhan, 659 (Vms.)
Patwardhan, V. N., with Chitre, 390 (Sp.)
Paul, B. M., with Pasricha & Panja, 580 (Chl.)
Paul, J. H., with Smithburn & Mahaffy, 416 (Misc.)
Paulraj, M., with Cochrane & Salmond, 27 (Lep.)
Payne, G. C. & Payne, F. K., 477 (Hel.)
Pearson, A. S., 693 (Fev.)
Peña Chavarria, A. & Kumm, H. W., 351 (Misc.)
Peney, M., 119 (Hel.)
Penna, R., with de Magalhães, 444 (Fev.)
Pereira, A., with Vaz, 657 (Vms.)
Pereira, C., 638 (Tryp.)
Perez-Vivas, M. A., with Gabaldon & Ochoa-Palacios, 562 (Mal.)
Pern, S., 663 (Vms.)
Perrin, T. L., with Lillie & Armstrong, 446 (Fev.)
Pesce, H., 24 (Lep.)
Pessôa, S. B. & Pestana, B. R., 264 *bis* (Leish.)
——, with Pestana, 264 (Leish.)
——, with —— & Corrêa, 264 (Leish.)
Pestana, B. R. & Pessôa, S. B., 264 (Leish.)
——, with Pessôa, 264 *bis* (Leish.)
——, —— & Corrêa, A., 264 (Leish.)
Phan-Huy-Quat, with Galliard & Dang-Van-Ngu, 45 (Hel.)
Philip, C. B., 443 (Fev.)
Philipsz, G. L. C., with Veeraraghavan, 159 (Rab.)
Phisalix, M., 167 (Vms.)
Pickels, E. G. & Bauer, J. H., 69 (Y.F.)
Pifano, F. & Dias, E., 639 (Tryp.)
——, with Mayer, 640 (Tryp.)
Pik Ging Hoo, 711 (Mal.)
Pinkerton, H. & Henderson, R. G., 533 (Misc.)
—— & Weinman, D., 355 (Misc.)
Pinotti, M., 409 (Mal.)
Piringer, W., 163 (Rab.)
Pisani, D., with Monacelli, 57 (Y. & S.)
Pizzillo, G., 32 (Mal.)
Pock-Steen, P. H., 13 (Sp.)
Pogodina, E. A. & Sokolov, A. G., 510 (Mal.)
Pons, L. M., with Niño & Gay, 729 (Misc.)
Porter, R. J. & Huff, C. G., 513 (Mal.)
Poynton, J. O., 153 (Hel.)
Pradhan, K. N. & Patwardhan, N. G., 659 (Vms.)
Prendergast, J. J., 465 (Lep.)
Proske, H. O., with Boyd, 563 (Mal.)
Public Health Reports, 164 (Rab.), 237 (Misc.), 623 (Pl.)
Puri, I. M., 359 (B.R.)
——, with Afridi, 105 (Mal.)

R.

Radaody-Ralarosy, P., 84 (Der.)
—— & Guidoni, P., 151 (Hel.)
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Raj, M. P., with Cochrane, Rajagopalan & Santra, 703 (Lep.)
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Ramirez, E., with Rivero, 568 (Mal.)
Ramsay, G. C. & Anderson, I. R., 232 (Mal.)
Randle, T. A., (233) (Mal.)
Rao, H. R., with Russell, 559 (Mal.)
Rao, P. K., 663 (Vms.)
Rao, R. S. & Ganapathi, K., 541 (Misc.)
Rao, S. R., 620 (Pl.)
Rao, S. S., 518 (Hel.)
—— & Maplestone, P. A., 152 (Hel.)
——, with Napier & Das Gupta, 521 (Hel.)
Rao, T. R., with Russell, 228, 642 (Mal.)
——, with —— & Knipe, 564 (Mal.)
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Ravenel, S. F., & Smith, D. L., Sr., 600 (Dys.)
Ray, H. N. & Idnani, J. A., 453 (Fev.)
Reardon, L. V., 588 (Am.)
Reed, A. C., 291 (Mal.), (423) (Misc.)
Regli, E., with Andreu Urra, J., & Andreu Urra, F., 573 (Leish.)
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Reiser, R., with Hanes, 389 (Sp.)
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Rennie, A. T., with Pakenham-Walsh, 231 (Mal.)
Reye, D., 350 (Misc.)
Reynes, V., 680 (Fev.)
Rhoads, C. P., with Erf, 389 (Sp.)
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Rhude, K., with Sawitz, d'Antoni & Lob, 523 (Hel.)
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Rita, G., 714 (Mal.)
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Roca-Garcia, M., with Bugher, Boshell-Manrique & Gilmore, 434 (Y.F.)
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Rodová, H., with Jirovec, 733 (Misc.)
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Rodriguez-Molina, R., 14, (14) (Sp.)
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Roe, M. A., with Kitchen & Huff, (36) (Mal.)
Røe, O., 18 (Sp.)
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Romiti, C., (153) (Hel.)
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Ross, A. O. F., 78 (Tryp.)
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Rothman, M. M. & Epstein, H. J., 594 (Am.)
Rottmann, A., 660 (Vms.)
Row, R., Ambegaonker, S. D. & Bhende, Y. M., 479 (R.B.F.)
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—— & Ganguly, S. K., 532 (Misc.)
—— & Ghosh, S. M., 531 (Misc.)
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Ruge, H., 199 (Fev.)
Ruiz, H., with Kumm & Komp, (352) (Misc.)
Ruiz-Castaneda, M., 687 (Fev.)
Russell, P. F., 410 (Mal.)
—— & Knipe, F. W. 645 (Mal.)
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——, with —— & Mohan, 646 (Mal.)
—— & Rao, T. R., 228, 559, 642 (Mal.)

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—— & Ruchman, I., 161 (Rab.)
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Sallam, H., 575 (Leish.)
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